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A STUDY OF THE ROENTGEN APPEARANCE OF CRANIAL VAULT SUTURES: CORRELATION WITH THEIR ANATOMY*

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RADIOLOGISTS are interested in the patency and configuration of the cranial vault sutures essentially for three reasons. The roentgenographic appearance of the suture may be mistaken for a fracture dehiscence. This has been discussed in a previous paper by one of us[13] and the locations that offer the most common pitfalls have been pointed out. The obliteration of the sutures may be pathologically accelerated, as in premature synostosis with its clinical and sometimes surgical consequences, or abnormally delayed as in cretinism and hydrocephalus. Finally, the state of the sutures offers a means of evaluating an aspect of physiologic differentiation and maturation throughout life and including ages where most other indicators of skeletal development have lost their usefulness.

REVIEW OF LITERATURE

Anatomists define cranial sutures as immovable joints in which the interlocking bones are connected by dense fibrous tissue. One group of investigators sees in the sutures the primary site of cranial growth, comparing them in this respect with the epiphyseal cartilages in the long bones.[3,15,29] Another group views the sutures essentially as a passive mechanism permitting slight movement between the individual bones and resisting gross separation caused by trauma or muscle pull.[5,20] According to Brash,[5] the appositional growth of the brain capsule is mainly periosteal in character, bone deposition on the outer surface of the skull being paralleled by bone resorption along the endocranial aspect. On the basis of extirpation experiments of sutural and periosteal areas of the rat cranium, Moss[17] presents a compromise aiming at reconciliation of the two conflicting views. He concludes that the suture is not the primary site of growth of the calvarial bones, rather their growth is a function of the periosteal osteogenic tissue surrounding all surfaces of the bones, ectocranial, endocranial, and in the sutural area. As the neurocranial capsule expands in response to the expansion of the brain, the bones within the capsule "float" out-

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ward, increasing the surface area by peripheral extension of their periosteal layers including extension into the presumptive sutural area. The latter is just one side of accretion of bone in the early stages of osteogenesis of the calvarial bones. The sutural extirpation experiments of Moss also prove that the site of the calvarial sutures in the growing rat is not predetermined and that the individual bones may extend beyond the normal location of the sutures. The position of the sutures thus is conditioned by the meeting of the bones arising from individual ossification centers. A recent anatomic and anthropologic study of Mednick and Washburn[16] distinguishes between thick skulled and thin skulled animal forms, the latter represented by man. The authors contend that in man the appositional and resorptive processes play a minor role in shaping the skull and that the relative growth potentials of the various vault sutures determine the final form to a greater extent than in any other animal. Apposition and resorption are responsible only for increasing the thickness of the bones and for the production of the sinuses and diploic spaces.

Radiology has made significant contributions to the understanding of cranial growth changes. Brodie[7] demonstrated by cephalometric determinations on serial roentgenograms of children's skulls that the growth of the cranium is essentially concentric and that the configuration of the skull is determined as early as the third month of life and thereafter does not change. Recently, Selman and Sarnat[21] again employed serial roentgenographic studies to determine sutural growth increments by measuring on the roentgenogram the separation of metallic implants on either side of the suture.

As a biologic phenomenon suture closure has puzzled scientists since the times of Hippocrates. Vesalius and his pupil Fallopius were familar with the fact that sutures undergo progressive obliteration with advancing age.[2] Numerous authors during the latter half of the last century and the early part of the twentieth century have interested themselves in different aspects of this problem. Recently, the literature has been surveyed again by McKern and Stewart.[14] Various studies have concerned themselves with sex and race differences in suture closure, order and variability of closure, and differentiation between endocranial and ectocranial closure. Most of the older authors came to the conclusion that suture closure is extremely irregular and for this reason is of little or no value for age determination.[14]

Todd and Lyon[23–26] have given the most definite information on the actual dates of suture closure and for the last thirty years their work has become the standard reference, also in radiologic textbooks and reference works. They subdivided each suture into smaller portions and assessed the state of fusion for each portion individually. They also assigned numerical values to indicate the degree of fusion from 0 for the open sutures to 4 for complete obliteration and arrived at dates for the beginning and completion of closure ectocranially and endocranially in white and Negro males. The voluminous studies of Todd and Lyon can be summarized as follows:

1. While endocranial and ectocranial suture closures start approximately at the same time, ectocranial closure progresses more slowly and more erratically.

2. Therefore, endocranial suture closure is a more reliable age indicator than ectocranial closure.

3. The greatest activity in suture closure occurs between the years of twenty-six to thirty. There are, however, secondary periods of obliterative activity in the late thirties (ectocranial), the fifties (endocranial), the sixties (ectocranial), and the late seventies (endocranial), which try to complete what has been missed during earlier spurts of fusion activity.

4. Endocranial fusion for the male white series is in the order of sagittal, coronal, lambdoid sutures.

5. While for a large series the average age computed from the state of the sutures

closely approximates the actual average age, the results in an individual case give a deviation of from six to ten years.

In recent years Todd and Lyon's work has been subject to criticism. Abbie[1] characterizes their studies as being "marred by arbitrary rejection of awkward material." Singer[22] points out that Todd and Lyon used only male skulls and eliminated all specimens which showed "irregularity of closure." He does not consider their explanation for "smoothing out" graph curves as valid.

Two impressive recent studies well summarize present day opinion. McKern and Stewart,[14] in a very scholarly report on identification of United States war dead from Korea, state that progress of suture closure has only a very general relationship with age. The onset and progress of clusure is so erratic that it cannot be relied upon for skeletal age identification. Brooks[8] concludes from her fair-sized series that as an age indicator in female skeletons cranial suture closure is of doubtful value; in males it should be utilized only as confirmatory to other indicators of adult age. The wide range in the age of suture closure even for the same area of the skull represents a challenge to the investigator to find the reasons for this great variability. Here, the roentgenologic approach is particularly useful since it can be applied to large numbers of cases which can be investigated simultaneously with the tools of the anthropologist, the clinician and his laboratory, and the psychologist. So far such correlations have not been made since most studies have been done on skeletal material where at best only information on age, sex, and racial origin was available.

Cross-sectional or horizontal studies for different age groups need to be undertaken as well as longitudinal studies which follow individuals through their life span by means of properly timed, but infrequent roentgenograms of the skull in one or two projections.[13] The radiation dosage could be restricted to a minimum and the gonads protected so that the obstacle of radiation hazard could be overcome.

The recent literature throws some light on the biology of suture closure. It has been known for some time that certain cranial malformations which are associated with premature obliteration of the sutures (synostosis), as *e.g.* dysostosis craniofacialis are hereditary. The uninterrupted occurrence of the latter disease in four generations suggests dominant inheritance.[4] In simple premature cranial synostosis opinions differ, but most authors agree that there also is a hereditary component involved. The primary cause is said to be a disturbance of the mesenchymal matrix in which the bone is laid down.[19] According to Torgersen,[27] the evidence is in favor of dominant genes with low penetrance being responsible for the malformation. On the basis of his genetic investigations, Torgersen comes to the conclusion that there are probably two groups of genes influencing the sutural pattern of the skull, one determining the site of the cranial ossification centers and location of the sutures, and another affecting the rate of suture obliteration. In a later paper Torgersen[28] calls attention to the great frequency of sutural variations at high altitude. This fact has been demonstrated for a number of different mountainous regions on several continents. While the occurrence may be of genetic origin, it can also point to the effect of some other selective factor, possibly of a metabolic nature, and could be related to the mineral and oxygen metabolism of the body. The late fusion of cranial sutures in cretinism exemplifies the hormonal influence on suture closure. Very little is known on the relationship of nutrition to obliteration of the sutures. Haas[11] quotes Russian sources which observed deviations during periods of famine.

Among local factors that affect suture closure, Caffey[9] mentions localized dural thickening which interferes with the normal stimulus of the growing and expanding brain. Moss[18] hypothesizes that both normal and abnormal sutural fusions are

functionally related to tensile forces transmitted to them by the fiber tracts of the dura mater. He demonstrates this by sectioning the rodent dural falx cerebri, which uniformly results in interference with normal endocranial fusion of the metopic (interfrontal) suture.

Almost thirty years ago Haas[10,11] suggested in two classical papers that the biologic process of suture closure should be investigated in the living by roentgenologic means and through serial investigations. He pointed to the shortcomings of pathologico-anatomic studies which cover only a transitory phasic aspect of suture biology and of animal experimentation on the sutures which creates artificial and not quite comparable conditions. He himself laid the foundation for roentgenographic sutural research in a monographic presentation of the subject in which he depicted the roentgenographic life history of the sutures in infancy and childhood and their obliteration at maturity. He called attention to normal variations in the roentgenographic appearance of the sutures and presented their pathology in osteogenesis imperfecta, hydrocephalus, premature synostosis, and imbecility. Without being able to fill the gap, he pointed to the ever recurring problem of sutural roentgen studies: the correlation of the roentgenologic findings with the underlying anatomy.

In the light of Haas' investigations, it should be kept in mind that the roentgen diagnosis of an open suture is based on the appearance of a translucency with sutural characteristics and in the proper location. The anatomic substrate of this appearance may be either a completely open or a partially closed suture; in the latter case we have to postulate the presence of sufficient unfused bone to make a dehiscence demonstrable, whether it is in the lamina externa, which is common, and/or in the diploë which is conceivable, or in the interna, which seems to be rare. Clinically, such a suture would be fused; anatomically and biologically it would be partially open.

It is the purpose of this paper to test by statistical means the reliability of the roentgen evaluation of the sutural status in the light of its anatomy.

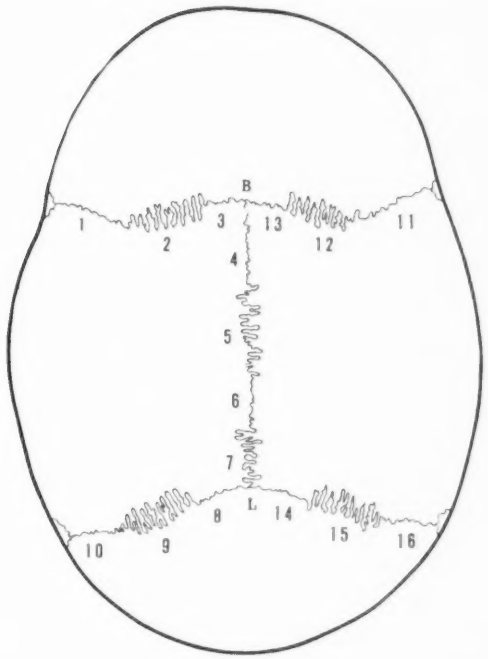
MATERIALS AND METHODS

Specimens for the study reported in this paper consisted of heads and calvaria from 26 dissecting room cadavers. Age, race, and sex of the specimens were known. In the sample there were 17 whites (15 males, 2 females), 8 Negroes (4 males, 4 females), and one Indian (male). Ages ranged from thirty-three to one hundred years. The majority (16) were in the sixth and seventh decade.

The cranial sutures were investigated both roentgenographically and by direct inspection after preparation of the head. The sutures were subdivided according to the original scheme of Broca[6] into 16 areas (Fig. 1).

Initially, roentgenograms were taken of the intact cadaver head. Anteroposterior, Towne, and lateral positions were utilized. Roentgenograms were made at a 48 inch target film distance, using 150 mas. and a kilovoltage commensurate with the thickness of the head varying from 70 to 90 kv. peak. Bucky diaphragm and par-speed screens were used in the anteroposterior and Towne positions. The Lysholm grid was employed in the lateral view because of difficulties in positioning the cadaver.

The areas illustrated in Figure 1 were examined on all roentgenograms on which they could be clearly discerned. In the lateral view, all areas of the coronal and lambdoid sutures were observed. In the anteroposterior view all areas were investigated. In this view, sagittal areas 4, 5, 6, and 7 were recorded as (a) an anterior portion adjacent to the coronal suture (to coincide with area 4), (b) a posterior portion adjacent to the lambdoid suture (to coincide with area 7), and (c) a middle portion between these two (to coincide with areas 5 and 6). The appearance of the sutural line was ascertained and recorded using the following classifications:



B – Bregma.
L – Lambda.

Coronal : 1 – 11 – pars bregmatica.
2 – 12 – pars complicata.
3 – 13 – pars pterica.

Sagittal: 4 – pars bregmatica.
5 – pars verticis.
6 – pars obelica.
7 – pars lambdica.

Lambdoid: 8 – 14 – pars lambdica.
9 – 15 – pars intermedia.
10 – 16 – pars asterica.

Fig. 1. Diagram of the subdivisions of the cranial vault sutures (after Broca).

(1) No line. This classification was recorded when no sutural line was observed (Fig. 2); (2) intermittent line. This classification was recorded when a clearly defined line was observed which followed an apparent sutural pattern but was not continuous (Fig. 3); (3) erratic line. This classification was recorded when a line was clearly evident, but did not seem to follow a sutural pattern; (4) continuous line. This classifi-

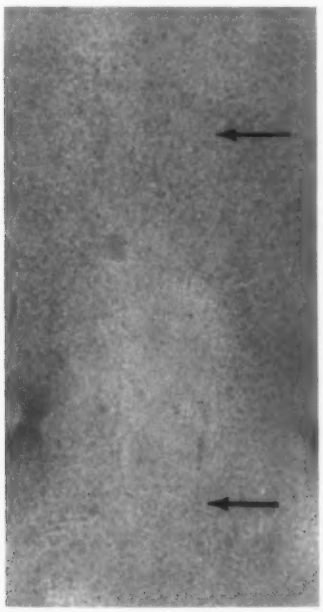


Fig. 2. Anteroposterior roentgenogram. No sutural line is observable in areas 5 and 6. (Area indicated between arrows.)

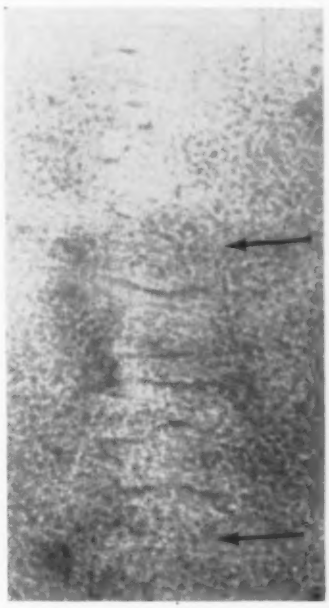


Fig. 3. Anteroposterior roentgenogram. An intermittent line is seen in area 7. (Area indicated between arrows.)

Fig. 4. Anteroposterior roentgenogram. Clearly defined, continuous suture line is evident in area 4. (Area indicated between arrows.)

cation was recorded when a clearly defined continuous sutural line was observed (Fig. 4); (5) fuzzy line. This classification was recorded when an apparent sutural line was present, but was not clearly defined. Due to difficulties in reproduction, classes 3 and 5 are not illustrated.

For anatomic evaluation the calvaria were removed from the cadaver in such a manner that the entire coronal and lambdoid sutures were retained. The periosteum and endosteum and dura were stripped from the calvarial bones and the sutures were investigated by direct inspection. All sixteen areas of the coronal, sagittal and lambdoid sutures as depicted in Figure 1 were recorded, both ectocranially and endocranially.

The extent of sutural closure was ascertained and recorded using the previously discussed and illustrated breakdown of each suture:[14,22,23,24] (1) Completely open sutural line (Fig. 5); (2) one-fourth of the sutural line closed (Fig. 6); (3) one-half of the sutural line closed (Fig. 7); (4) three-fourths of the sutural line closed (Fig. 8); (5) complete closure of the sutural line (Fig. 9). In classes 2, 3 and 4 no distinction was made between continuous and intermittent closure.

RESULTS

Table 1 shows the observations made on roentgenograms of the intact cadaver heads, as compared with observations of

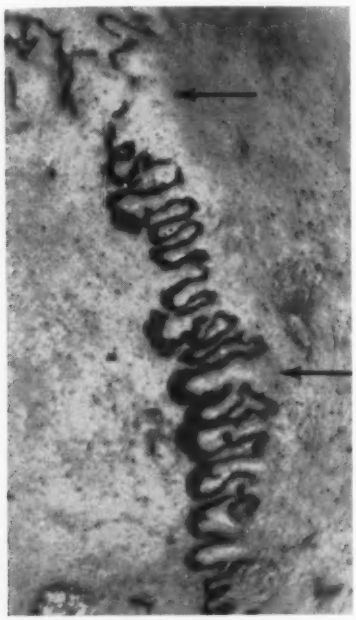


Fig. 5. Completely open suture is seen in area 8 on cadaver calvarium. (Area indicated between arrows.)

Fig. 6. One-quarter fusion is present in area 7 on cadaver calvarium. (Area indicated between arrows.)

the ectocranial and endocranial suture closure made by direct inspection of their calvaria. All observations made, whether in the anteroposterior or lateral view, are shown for all 16 of the sutural areas defined. In order to avoid minor classifications, one-quarter and one-half closures have been combined in tabulation, as have three-quarter and full closures. It will be seen that anatomically open sutures are in the minority, as would be expected in the age group represented by the cadavers. Many more sutures are open ectocranially than endocranially, and in only 8 cases is the ectocranial closure appreciably more advanced than the endocranial closure. Partial closure (one-quarter to one-half) is rarer in endocranial than in ectocranial closure, indicating a more rapid course for endocranial closure.

Sutures still open, both endo- and ectocranially, all show roentgenographically either intermittent or complete lines, with complete lines in slightly greater numbers. Apparently, a suture which is completely open on the two visible surfaces does not always show a continuous line on the roentgenogram. Surprisingly, where beginning closure is occurring on the endocranial surface, a complete line is more often seen than when the suture is apparently open on both surfaces.

Fig. 8. Three-quarters fusion is present in area 7 on cadaver calvarium. (Area indicated between arrows.)

Sutures still open ectocranially but nearly or fully closed endocranially—a large group—may show any roentgeno-

Fig. 7. One-half fusion is present in area 4 on cadaver calvarium. (Area indicated between arrows.)

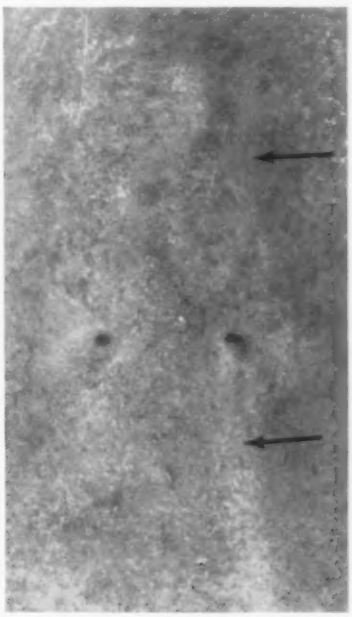


Fig. 9. Suture is completely fused in area 6; no line can be seen on cadaver calvarium. (Area indicated between arrows.)

TABLE I

OBSERVATIONS BASED ON A SINGLE ROENTGENOGRAM (ANTEROPOSTERIOR OR LATERAL) OF INTACT CADAVER HEAD

Ectocranial Suture Closure Seen in Denuded Skull	Open			One-quarter to One-half Closed			Three-quarters to Fully Closed			Total
Endocranial Suture Closure Seen in Denuded Skull	Open	One-quarter to One-half Closed	Three-quarters to Fully Closed	Open	One-quarter to One-half Closed	Three-quarters to Fully Closed	Open	One-quarter to One-half Closed	Three-quarters to Fully Closed	
Roentgenographic Appearance of Suture in Intact Body										
No line	0	1	86	0	1	30	0	0	93	211
Erratic or fuzzy line	0	0	29	0	0	21	0	0	4	54
Intermittent line	25	2	29	0	0	44	0	0	10	110
Complete line	30	12	43	0	1	13	0	6	12	117
Total	55	15	187	0	2	108	0	6	119	492

graphic appearance from "no line," which has a considerable plurality, to a "complete line." Erratic and fuzzy lines make their appearance in this class. Since endocranial closure is evidently well in advance of ectocranial closure in nearly all cases, the changes after this stage are in the degree of ectocranial closure. Passing from the third to the sixth column we see the change which appears when the ectocranial suture begins to close. Complete lines decrease, intermittent lines increase, erratic and fuzzy lines maintain the same proportional frequency, and the relative frequency of "no line" becomes less. The last effect is a reversal of the expected trend and again indicates that with beginning closure, ectocranial as well as endocranial, there is an actual roentgenographic "opening-up." An explanation for this phenomenon will be attempted later on in this paper. In the final state, closure nearly or fully complete ectocranially as well as endocranially, "no line" becomes the predominant class. It must be noted, however, that even in this group intermittent or even complete lines may still occasionally appear on the roentgenogram.

Table I gives the expectations of osteologic findings if the readings were based on only a single view (anteroposterior or lateral). Since in actual practice two or more views of the head of a given individual are taken to judge the condition of the suture, Table II has been prepared. In this table each sutural area of a skull has been classified according to the most distinct line which was found in either of the two roentgenograms taken. Thus, unfavorable views in which a sutural line may be partly concealed or distorted due to the angle at which the roentgenogram was taken are discounted as far as possible. The numbers in this table are smaller since here each individual head can be tabulated only once, whereas in Table I each subject may appear twice if the observation was attempted on both of the roentgenograms. In general, as must be expected from the method of classification, this table shows a higher relative frequency of better visualized lines; as, for instance, the percentage of cases with "no line on either roentgenogram" is less than the percentage of all "no line" observations in the first table. In general, however, no change need be made in the conclusions drawn. Even when two views are considered, there still are some cases in which no more than an intermittent line is seen on the roentgenogram although the suture is apparently open, both ectocranially and endocranially. When the endocranial line begins to close, the ectocranial line still being open, the incidence of "complete line" increases slightly. Cases in which the endocranial line is closed and the ectocranial line open still show a complete gamut of roent-

TABLE II

OBSERVATIONS CLASSIFIED BY MOST DISTINCT LINE SEEN EITHER ON ANTEROPOSTERIOR OR LATERAL ROENTGENOGRAM OF AN INTACT HEAD

Ectocranial Suture Closure Seen in Denuded Skull	Open			One-quarter to One-half Closed			Three-quarters to Fully Closed			Total
Endocranial Suture Closure Seen in Denuded Skull	Open	One-quarter to One-half Closed	Three-quarters to Fully Closed	Open	One-quarter to One-half Closed	Three-quarters to Fully Closed	Open	One-quarter to One-half Closed	Three-quarters to Fully Closed	
Roentgenographic Appearance of Suture in Intact Body										
No line on either film	0	0	30	0	0	7	0	0	28	65
Erratic or fuzzy line	0	0	14	0	0	7	0	0	4	25
Intermittent line	7	1	27	0	0	30	0	0	9	74
Complete line on one or both films	22	7	33	0	1	13	0	4	10	90
Total	29	8	104	0	1	57	0	4	51	254

genographic appearances. Paradoxically enough, it is again apparent that when ectocranial closure begins (the endocranial line in such cases being almost invariably already closed) the percentage of more clearly defined lines on the roentgenogram actually increases. In the last category of cases, in which the sutures are three quarters or more closed, both ectocranially and endocranially, the frequency of "no line on either roentgenogram" is smaller than the crude frequency of "no line" observations. Intermittent or even complete lines, it may be noted again, can appear on the roentgenogram though the sutures are nearly or fully closed to gross observation on their surface aspects.

In Figure 10 a graphic summary of Table 1 is given. Divisions along the X-axis are in proportion to the number of cases in various classes as divided by combinations of ectocranial and endocranial suture closure. The two very small classes in which endocranial closure is more advanced than ectocranial closure have been omitted. It is clear that the increase of "no line" and decrease of "complete line" on the roentgenogram does not progress uniformly with advancing stages of osteologically observed closure. The irregularity in the

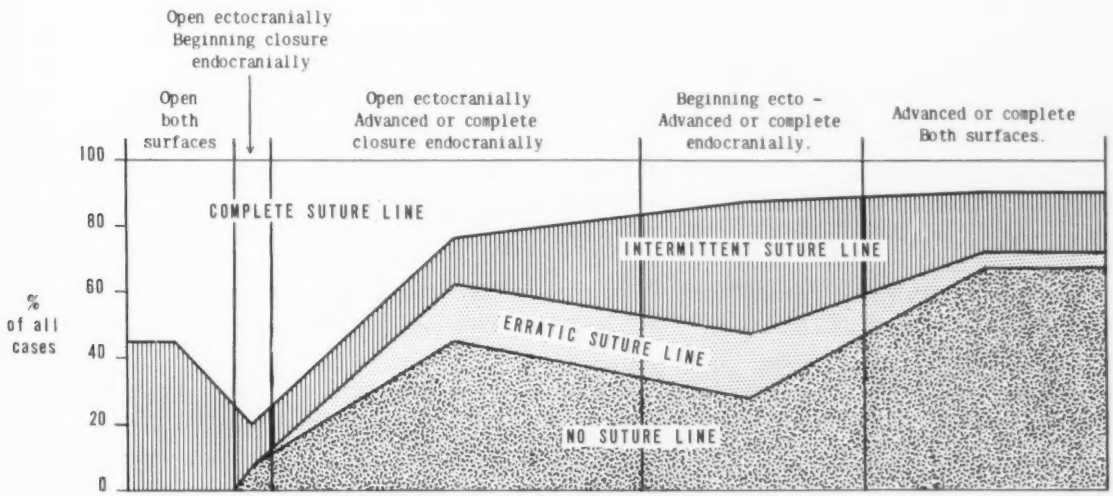


FIG. 10. Changes in roentgenologic appearance of cranial sutures with progressive stages of closure.

TABLE III

OBSERVATIONS IN CASES WHERE THE ROENTGENOGRAPHIC APPEARANCE WAS IDENTICAL IN BOTH ANTEROPOSTERIOR AND LATERAL VIEWS OF AN INTACT HEAD

Ectocranial Suture Closure Seen in Denuded Skull	Open			One-quarter to One-half Closed			Three-quarters to Fully Closed			Total
Endocranial Suture Closure Seen in Denuded Skull	Open	One-quarter to One-half Closed	Three-quarters to Fully Closed	Open	One-quarter to One-half Closed	Three-quarters to Fully Closed	Open	One-quarter to One-half Closed	Three-quarters to Fully Closed	
Roentgenographic Appearance of Suture in Intact Head										
No line	0	0	30	0	0	7	0	0	28	65
Erratic or fuzzy line	0	0	4	0	0	2	0	0	0	6
Intermittent line	4	0	2	0	0	9	0	0	0	15
Complete line	8	5	10	0	0	0	0	2	2	27
Total	12	5	46	0	0	18	0	2	30	113

second block is not statistically significant because of the smallness of the group. However, in the group "beginning ectocranial closure and advanced endocranial closure," there is a statistically significant increase in visible lines although the reverse would be expected. Suture lines occur in this class with significantly higher frequency than in the preceding group, in which the ectocranial portion is entirely open.

Table III shows a tabulation of only those cases in which the roentgenologic observation was the same in both lateral and anteroposterior views. This rather select group, too small for much statistical evaluation, shows a relative dearth of the intermediate (intermittent, erratic) classifications which were apparently less likely to be made consistently in both views. On the whole the correlation shown in this chart is superior to those previously shown, though considerable irregularity in the relation of roentgenologic and gross appearance is still present. The essential findings of the other tables are borne out.

DISCUSSION AND CONCLUSIONS

Relatively early in the development of his field, the radiologist accepted the fact that complete agreement betwen roentgenologic readings and direct anatomic inspection could not be expected. Recently, Keen[12] confirmed this by stating that anatomic and roentgenographic criteria for age determination cannot be reconciled and must be treated as separate entities. The differences in results were generally explained on the basis of differences in methodology, with direct anatomic inspection being regarded as superior in revealing anatomic detail. Haas[10,11] seems to have been the only one who undertook comparative studies of the calvarial sutures on roentgenograms and their anatomic counterparts. As his study object he used macerated skulls from a museum collection. Haas came to the conclusion that the roentgenographically demonstrable suture gap corresponds to its ectocranial portion, except for a few favorable cases in which the endocranial part is projected as a straight or slightly wavy line into or adjacent to the ectocranial suture. So far as is known no comparative studies have been made which correlate qualitatively and quantitatively the roentgenologic findings on cranial sutures of the intact head with the results of direct anatomic observations on the ecto- and endocranial aspects of the same calvaria after removal of all soft tissues. Our report tries to fill this gap.

The results of our comparative studies were found to be quite complex. It can be stated with near certainty that if no suture line is seen on the roentgenogram, the endocranial suture is at least three-quarters closed. There were only two exceptions to this rule out of 211 observations. In these

two instances the suture was closed only one-quarter to one-half on its endocranial aspect. This result is of considerable practical importance, particularly in the roentgen diagnosis of premature synostosis where, in the light of our observations, the absence of a suture line on the roentgenogram denotes a clinical fusion of the suture. It must be remembered, as previously pointed out that, in clinical terms, if a suture is closed, even only on its endocranial aspect, it means fusion.

Our results further show that a suture which is open anatomically on its outer and inner aspects is invariably demonstrable on the roentgenogram either as an intermittently open line or more commonly as a completely open line. If the suture is closed endocranially but open ectocranially, all variants of roentgenographic findings occur from disappearance of the suture line to complete patency.

Unexpected and thought provoking is the frequent appearance of intermittently or completely open roentgenographic suture lines in skulls, where both ecto- and endocranial surfaces were three-quarters to fully closed. Related to this and paradoxical seems the increase in frequency of roentgenographically open suture lines when we compare skulls with ecto- and endocranially open sutures with crania that display beginning endocranial fusion. This surprising phenomenon of increasing suture visibility is statistically confirmed for those cases where beginning closure ectocranially is combined with a closing or closed endocranial suture. Our observations seem to indicate that progressive stages of both ecto- and endocranial closure are temporarily accompanied by changes that result in a roentgenographic "opening up" of the suture. What does this mean in anatomic terms? It is conceivable that suture closure on the ecto- and endocranial aspects is accompanied by bone absorption in the diploë. This would explain the increasing roentgenographic visibility of the suture line in the presence of advancing surface closure. Another possibility is that the plane of the suture, which often extends at an oblique angle from the external to the internal aspect of the cranium, assumes a right angle in relation to the surface as the suture begins to close. This also would result in increasing roentgenographic visibility of the suture gap in the diploë.

From our observations it can be concluded that the roentgenographic dehiscence is neither the resultant of the ectocranial gap only, as the literature supposes, nor of the diploic or endocranial aspects of the suture, but a composite of all three with varying shares of each. This would also explain why sutures, which are open ectocranially but nearly or fully closed endocranially, may offer any roentgenographic appearance from absence of a suture line to the intermittently patent or completely open line. It should be realized here that the dentations of a suture are not just surface characteristics, as so often assumed, but extend through the depth of the suture into the diploë and beyond. Even the inner (dural) aspect of the suture may show more serrations than it is generally given credit for. If the suture line seen on the roentgenogram is considered as a two dimensional projection of a three dimensional space, the variation in the roentgenographic image of the suture in spite of identical surface appearance is more easily understood. This explanation of our observations will have to be tested by various means, but particularly by histologic and microradiographic techniques. Such studies are in progress. It is conceivable that the roentgenologic appearance of a suture which apparently is based on its total cross section may give a better indication of its status for purposes of anthropologic, physiologic, and age evaluation than the anatomic appraisal of only its surface aspects.

SUMMARY

1. In addition to representing a pitfall in the diagnosis of skull fractures, the roentgen appearance of cranial vault sutures is of interest to the radiologist because (a)

it furnishes important evidence in the diagnosis of abnormal acceleration or delay of sutural fusion and (b) it offers a means of evaluating physiologic maturation throughout life and at ages where other indicators of skeletal differentiation have lost their usefulness.

2. In order to test the reliability of the roentgen evaluation of the sutural status, a comparative study was made which correlates the roentgenologic findings on the vault sutures of 26 intact cadaver heads with the results of direct observation on the external and internal aspects of the calvaria of the same heads after removal of all soft tissues.

3. It was found that if no suture line is seen on the roentgenogram, the endocranial suture is at least three-quarters closed.

4. A suture which is open anatomically on its outer and inner aspects is invariably demonstrable on the roentgenogram, either as an intermittently open line or more commonly as a completely open line.

5. If the suture is closed endocranially but open ectocranially, all variants of roentgenographic findings occur, from disappearance of the suture line to complete patency.

6. Frequently and surprisingly, intermittently or completely open roentgenographic suture lines were visible in skulls in which both ecto- and endocranial surfaces were three-quarters to fully closed.

7. Roentgenographically open suture lines seem to increase in frequency when calvaria with ecto- and endocranially open sutures are compared with crania displaying beginning endocranial fusion.

8. Statistically proved is the paradoxical increase in suture visibility when we compare skulls with endocranial fusion and ectocranial patency with skulls in which endocranial fusion is combined with beginning closure of the ectocranial aspect.

9. It is suggested that this roentgenographic "opening-up" of the suture may be explained by (a) bone absorption in the diploë occurring with suture closure of the ecto- and endocranial surface aspects or (b) the plane of the suture assuming a more favorable angle for visibility as the suture begins to close. Either of these possibilities would result in increasing visibility of the sutural gap in the diploë.

10. The dentations of a suture extend through the depth of the suture into the diploë and beyond. Even the dural aspect of the suture shows more serrations than is generally believed. It is suggested that the roentgenologic appearance of a suture is based on its total cross section and that it therefore may give a better indication of the sutural status for purposes of anthropologic, physiologic, and age evaluation than the anatomic appraisal of only its surface aspects.

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ROENTGENOLOGIC MANIFESTATIONS OF FRACTURE OF THE ORBITAL FLOOR (BLOW-OUT FRACTURE)*

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THE incidence of depressed fractures of the orbital floor is probably greater than many radiologists realize. These important injuries are frequently overlooked in cases of facial trauma because of the observer's lack of awareness that such fractures may be present although the orbital rim itself is intact. It is believed that the diagnosis of orbital floor fractures would be made with greater frequency if there was more general knowledge among radiologists of the etiologic mechanism involved. Familiarity with the roentgenologic manifestations is essential as well as a better realization of the need to obtain a carefully planned roentgenographic examination.

Pfeiffer's[1] article on traumatic enophthalmos was a comprehensive review on this subject and one of the first to appear in the American medical literature. More recently, Smith and Regan[2] described the mechanism and correction of internal orbital fractures. It seems unlikely, however, that either of these excellent papers received any widespread publicity among radiologists since they appeared in ophthalmologic literature. Most standard American textbooks on diagnostic radiology make no mention of this particular phase of facial trauma, the unique exception being that of "The Skull, Sinuses, and Mastoids" by Young,[3] who described it briefly but thoroughly and illustrated one example.

The expression "blow-out" or hydraulic fracture as used by Smith and Regan[2] seems particularly expressive in referring to a fracture of the normally extremely thin floor of the orbit. This type of fracture is caused by a sudden increase in intra-orbital pressure occurring when an object hits the front of the orbit, and, with or without rupturing the eyeball, forces the orbital contents backwards. In this type of injury, the thin medial orbital wall may also be fractured, or occasionally the medial wall alone is fractured. The degree of depression of orbital floor fragments into the antrum appears to be related not only to the force of the injury but also to the antral size. Although Smith and Regan produced this injury experimentally in the cadaver using a smooth hurling ball, it is mentioned by them and others that blows from fists as well as smooth rounded objects likewise may produce it. In the present authors' series blows from clenched fists lead by far in being the chief offender in the production of this injury.

REPORT OF CASES

Case I. A forty-year old female was struck in the left eye with a fist. She was seen two days later with severe edema, ecchymosis and chemosis of the left eye and lids, moderate proptosis and limited motility. The visual acuity in the involved eye was reduced to 20/30. The remainder of the examination was negative as far as could be determined. She was treated conservatively with compresses and mydriatics. On the second visit, four days later, some of the edema and proptosis had subsided and at this time limitation of motility in the fields of action of the superior and inferior rectus muscles was noted. Roentgenograms (Fig. 1, *A* and *B*) revealed fragmentation of the floor of the left orbit and depression of these fragments into the upper left maxillary sinus. On the third visit, there was further reduction in the edema and at this time diplopia and weakness of the left superior oblique muscle were noted. The visual acuity of this eye was still 20/30. She was referred to the otorhinolaryngology service for treatment of the depressed fracture of the orbital floor.

Case II. A thirty-eight year old male was struck in the left eye and orbit by a board. He

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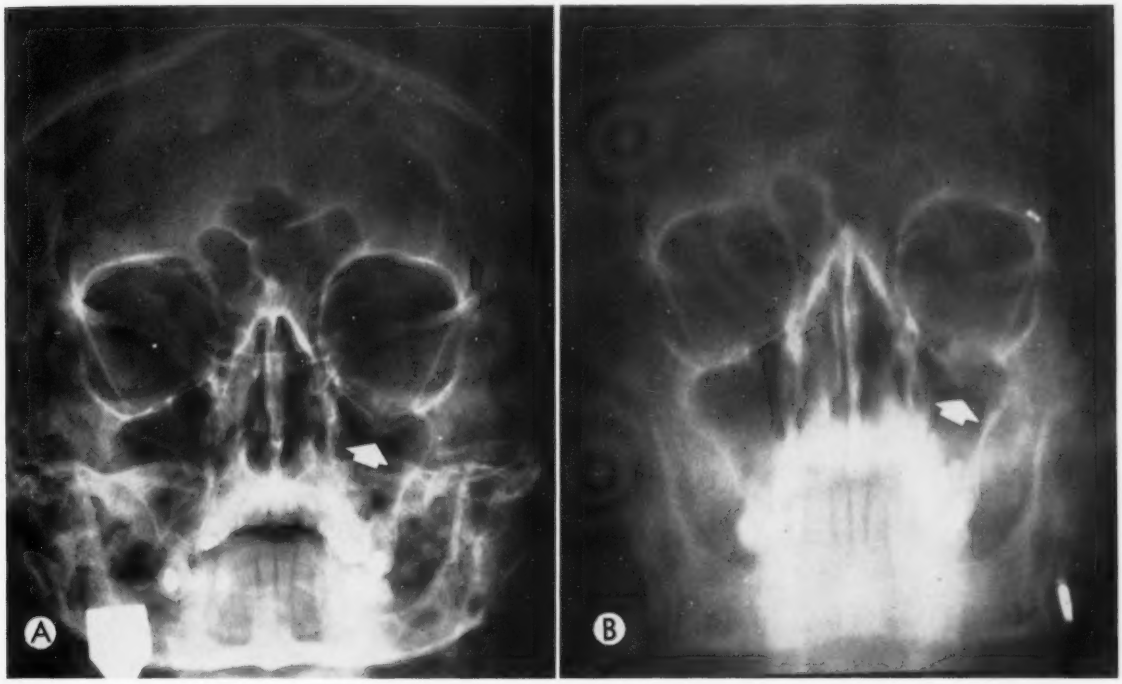

Fig. 1. (*A* and *B*) Depressed fracture of left orbital floor with characteristic appearance (arrows). Optimal tilt of head reveals abnormality to best advantage in both conventional roentgenogram (*A*) and laminagram (*B*).

was seen the next day with tense swollen ecchymotic lids, and marked proptosis of the left eye which was essentially immobile and directed upward. He had a moderate traumatic iritis and his vision was reduced to about 20/100 in the involved eye. Roentgen examination that night was believed not to reveal any bone injury but there was fluid noted in the left antrum. The local swelling and proptosis subsided somewhat and subsequent ocular examination disclosed retinal edema in the macular region. Because of the persistent left hypertropia and vertical diplopia, supplementary roentgen examination was performed on the seventh day and this latter study indicated that an orbital floor fracture was present. The patient was referred to the otorhinolaryngology service for reduction of the fracture. A Caldwell-Luc procedure was performed during which the orbital floor was elevated and the antrum packed. Following surgery, the motility of the inferior rectus muscle improved and the degree of left hypertropia was reduced. However, it is anticipated that in the future the floor of the orbit will have to be explored and further vertical muscle surgery performed.

Figure 2, *A* and *B* illustrates the easy obscuration of the pathology by failure to obtain the critically correct modification of the Waters projection. These roentgenographic studies were performed one and seven days, respectively, following injury and without the knowledge of the ophthalmologic abnormalities present. They emphasize the importance of individualizing the roentgenologic examination and how exaggeration of the backward head tilt (Fig. 2*A*) or a more vertical positioning of the head (Fig. 2*B*) may make roentgenologic assessment difficult or impossible. Figure 2, *C* and *D* is a roentgenogram in a correctly modified Waters view and a laminagram (4 cm. from table top), respectively, in the same case.

Case III. A twenty-eight year old female was struck in the right eye by a bottle. She was seen immediately after the injury at which time she had mild blurring of vision in the right eye, a 4 cm. laceration of the skin of the right upper lid, hematoma of the lids with mild proptosis and extensive subconjunctival hemorrhage. Ocular motility was good; there was evidence of a mild iridocyclitis, but the fundus was normal. Roentgen examination (Fig 3, *A* and *B*) demonstrated a depressed fracture of the floor of the right orbit and, in addition, an area of interstitial emphysema was noted

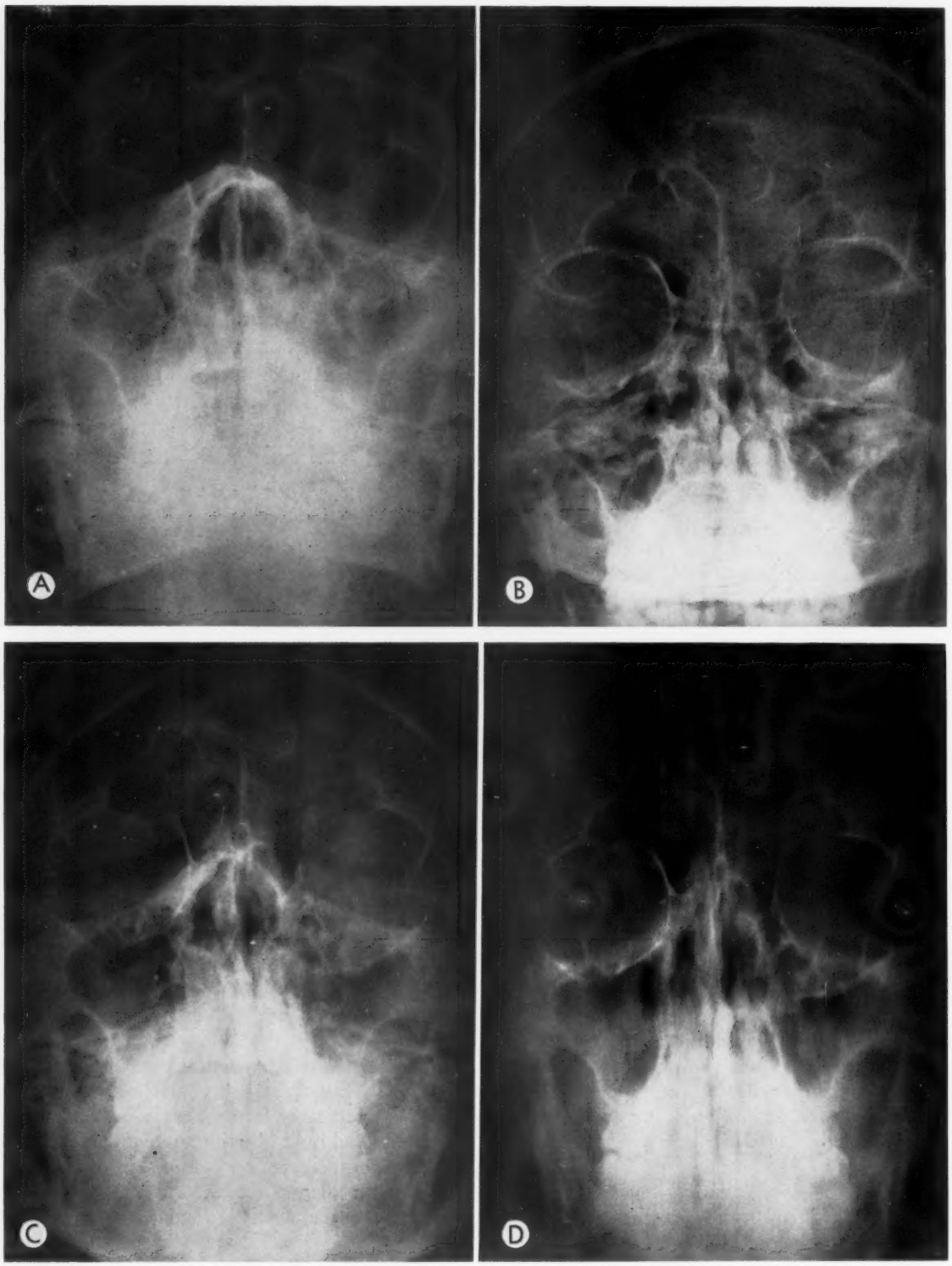

Fig. 2. (*A–D*) Left orbital floor fracture demonstrated by roentgenographic and laminagraphic studies, using the modified Waters position. Correct positioning (*C* and *D*) clearly shows depression of floor, in contrast to poor visualization in the exaggerated Waters and Caldwell views (*A* and *B*).

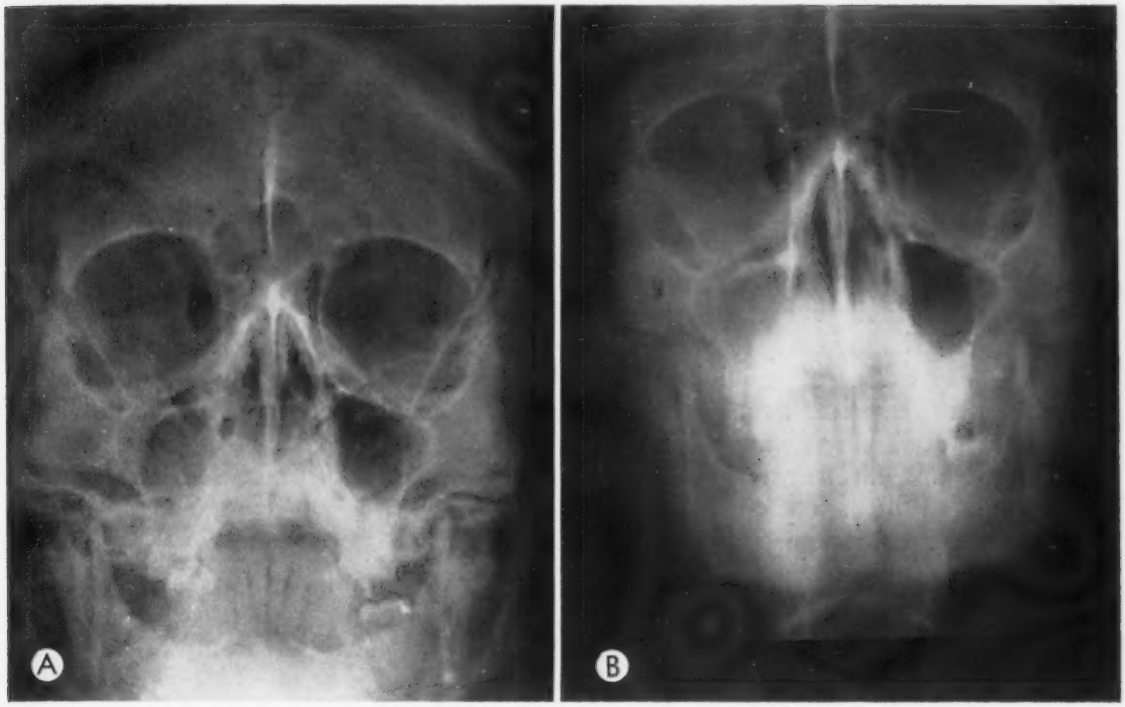

Fig. 3. (*A* and *B*) Depressed fracture of right orbital floor. Hemorrhage into antrum. Interstitial emphysema within orbit suggests accompanying fracture of medial orbital wall (*A*). Laminagraphic corroboration (*B*).

within the orbit and lateral to the medial orbital wall, indicating that there probably also existed a fracture of the medial orbital plate. The patient was admitted to the otorhinolaryngology service and a reduction of the fracture with antral packing was performed.

DISCUSSION

Clinically, fractures of the orbital floor should be suspected after any blow to the eye which produces a severe ecchymosis of the eyelids. There may be intra-ocular damage or there may be a contusion injury to the eye severe enough to produce permanent loss of vision. Occasionally, enophthalmos is present initially; usually there is a retrobulbar hemorrhage which produces proptosis of the eye and limitation of ocular motility. After reabsorption of the hemorrhage, the eye characteristically becomes enophthalmic. Permanent diplopia will result if the eye also becomes depressed or if the inferior rectus or inferior oblique muscle is incarcerated in bone fragments or in secondary scar tissue. In the latter case, the eye movement will be limited in both elevation and depression. Prompt diagnosis is important since early surgical reduction of the fracture will reduce the likelihood of permanent diplopia and will prevent a disfiguring degree of enophthalmos. If there is a significant depression of the orbital floor in an early case, the fragments may be elevated through a Caldwell-Luc type of antrotomy and held in place with antral packing. Smith and Regan[2] advocate early exploration of the orbital floor with grafting of autogenous iliac bone. We prefer to reserve this procedure for delayed cases or cases in which there is no retrobulbar hemorrhage and there is obvious incarceration of the inferior rectus muscle. The presence of proptosis and hemorrhage makes exploration or evaluation difficult. In most cases with proptosis the inferior rectus and inferior oblique muscles are stretched and pulled free of the fracture.

With or without fractures of the rim, the floor of the orbit to a varying extent be-

comes depressed into the antrum, but, unfortunately, this alteration may be overlooked roentgenologically for a multiplicity of reasons including: ignorance on the part of the observer that orbital floor fractures can be present even though the orbital rim is intact; obscuration of the detail of the antrum by accompanying hemorrhagic extravasation into it at the time of injury; and failure to obtain a roentgenographic examination optimally designed to demonstrate the full extent of the injury. The orbital floor is visualized as a slender curvilinear osseous density depressed into the attic of the antrum to varying degrees. Just superior to the displaced osseous fragment or fragments may be visualized a soft tissue density representative of downward prolapse of the edematous inferior orbital contents. Most authors agree that the Waters and Caldwell views are those of chief value for the roentgenographic delineation of this injury but it is reported by some that laminagraphy is of little value in demonstrating it. It is our opinion that a carefully individualized roentgenologic examination with this entity in mind and a high index of suspicion in all eye injuries will appreciably increase the incidence of correct roentgenologic diagnosis. It would appear from our own experience that a selectively modified Waters projection is the roentgenologic examination of choice but supplementary laminagraphy utilizing this chosen modification is helpful when soft tissue swelling interferes with visualization.

From the roentgen standpoint examination is probably ideally deferred until the periorbital soft tissue swelling subsides, this delay allowing the frequently concomitant intra-antral hemorrhagic extravasation to become absorbed whereupon the regained air content of the antrum will enhance contrast and therefore roentgenographic definition. However, customarily these injured patients are studied roentgenologically soon after the trauma is incurred, and since there is some urgency relative to making a definitive diagnosis all possible methods of arriving at a conclusive decision should be utilized without undue delay. In spite of the presence of obscuring densities in the area resulting from hemorrhage into the antrum and soft tissue swelling, it is believed that most cases of orbital floor injury can be identified roentgenographically, providing the interpreter is aware of the entity and is supplied with the suitable film examination.

SUMMARY

The roentgenologic manifestations of depressed orbital floor fractures without demonstrable orbital rim injury have been described and illustrated by several proved cases. Carefully individualized roentgenologic examinations utilizing a modified Waters projection and laminagraphy appear to be definite aids in the necessarily early diagnosis of this important and increasingly common type of facial injury.

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THE CLASSIFICATION OF CERVICAL SPINE INJURIES

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AN ANALYSIS of the mechanism of cervical spine injury is necessary for complete understanding of the clinical, roentgenographic, and pathologic findings of such cases. We propose a classification based on the dynamics of injury, attempting to correlate all these factors.

Barnes[1] made a valuable contribution to the study of mechanism by demonstrating that flexion injuries to the cervical cord are not possible without complete dislocation of one or both of the inferior articulating facets with locking against the leading edges of superior facets of the vertebra below. Taylor and Blackwood[5,6] described damage of the cervical cord by posterior dislocation secondary to hyperextension of the neck. Neither these mechanisms nor the time-honored explanation of acute traumatic disk herniation were found to explain all the cases encountered in our series. We have found it necessary to study other possible mechanisms of cervical spine injury.

I. FLEXION INJURIES

Flexion injuries are those usually caused by blows received to the posterior aspect of the skull, forcing the face toward the chest. A stress is placed upon the ligamentum nuchae, the interspinous ligaments, and the other posterior ligaments of the neck. If the force is powerful enough, severence of the posterior ligamentous structures may occur with varying degrees of forward dislocation of the facets at the level where the break occurs. The most severe form of this type of injury occurs when after complete dislocation, anteriorly, one or both of the dislocated facets lock. Under such circumstances, the neural elements will be contused between the posterior superior aspect of the vertebral body below the dislocation and the lamina of the vertebra above.

The stress imposed on the posterior ligaments may cause fractures of the posterior elements of the vertebrae as they are forced apart (principally the spinous process but occasionally a lamina or pedicle). If there is significant dislocation, a stripping of the anterior longitudinal ligament from the vertebra below will occur which may pull a chip from the anterior superior surface of the vertebral body below (Fig. 1 and 2, *A–F*).

A. *Flexion without Compression.* If the injuring force has a forward and upward component, the primary injury involved will be ligamentous. There may or may not be fractures of the posterior elements, dislocation of the facets, and an anterior chip.

Roentgenographically, the injury may present as a partial dislocation of the facets or complete dislocation with locking of both of the facets. If dislocation does not occur, or if it is reduced, the injury may be evidenced only by a widening of the posterior aspect of the intervertebral space with widening of the distance between two spinous processes. No bony evidence of injury may be present, and lateral flexion views may be required to demonstrate ligamentous injury.

Unilateral flexion injuries are caused by a blow delivered to the lateral aspect of the posterior skull turning the face to the opposite side. (The position of the head and angle of the blow may complicate this simple example.)

Roentgenographically, there may or may not be gross abnormal curvature of the cervical spine. The posterior ligamentous structures will be severed at least on the same side as the fracture. The facets will be unilaterally dislocated and locked, or the superior articulating facet below the ligamentous break will be fractured. The

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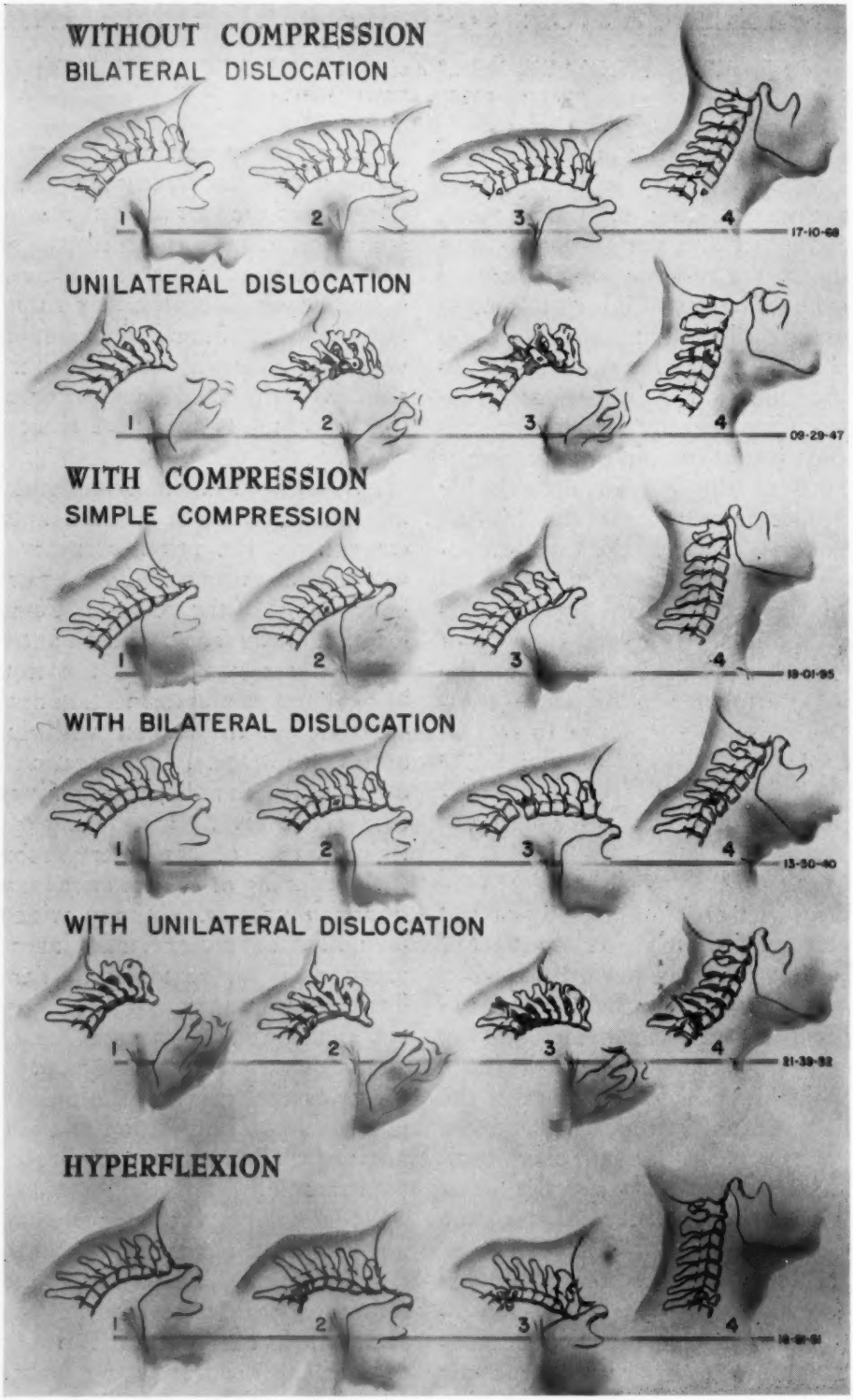

FIG. 1. *Flexion Injuries.* Each line of drawings illustrates the dynamics of a single type of injury (drawings 1 through 3) and a typical end result (4).

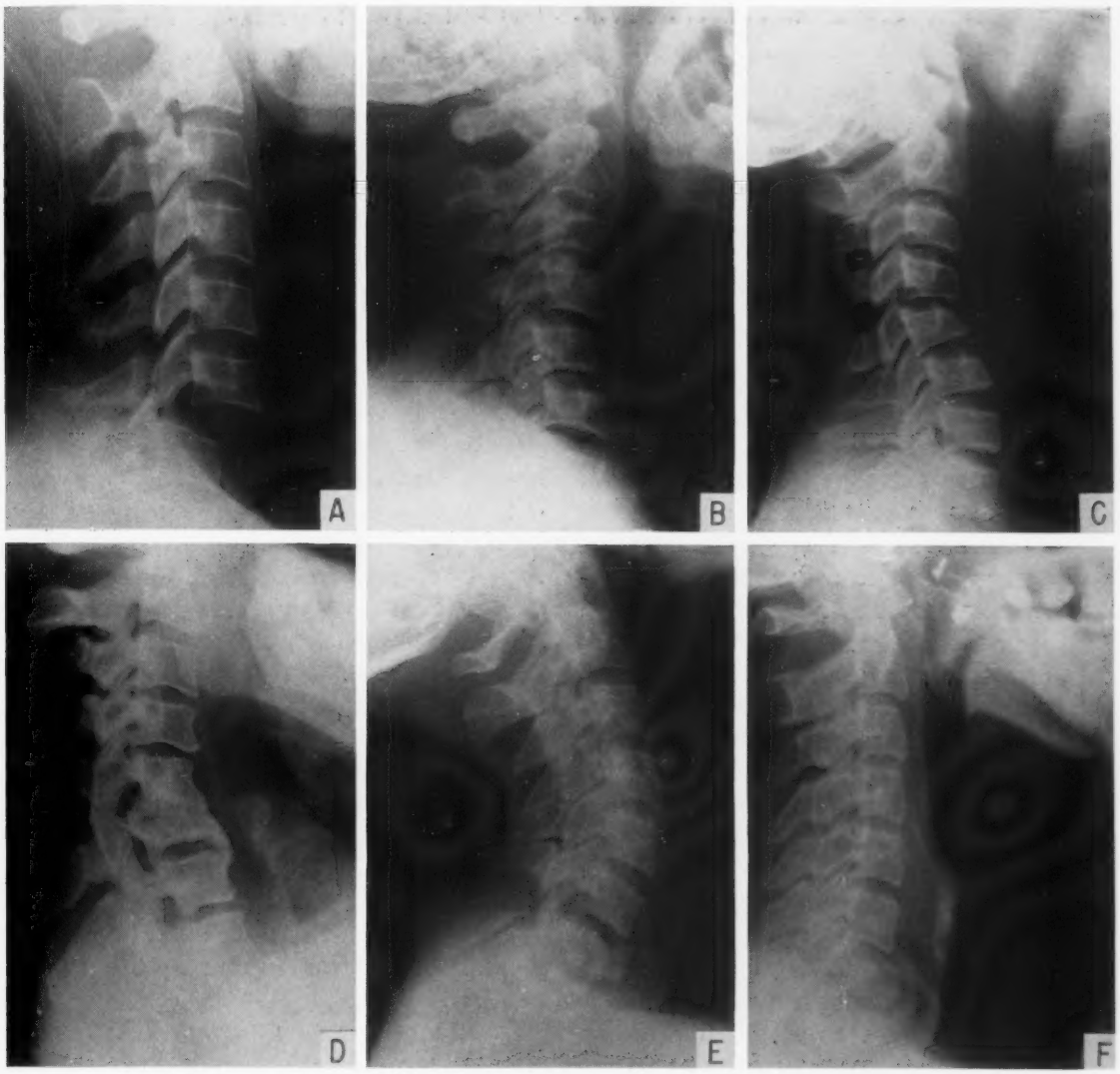

Fig. 2. The actual cases of *flexion injuries* illustrated in Figure 1. (*A*) 17–10–68. Thirty-nine year old male fell from a roof striking the occiput. Immediate paralysis of the lower extremities without loss of consciousness. Bilateral flexion injury without compression illustrating locked facets and an anterior chip torn away by the anterior longitudinal ligament. (*B*) 09–29–47. Thirty-four year old male thrown from a horse. Paraplegia. Unilateral (rotary) flexion injury without compression, demonstrating a single locked facet, rotary dislocation of the vertebrae, and a fractured spine secondary to ligamentous strain. (*C*) 19–01–95. Seventeen year old male struck his head on the bottom of a sandy lake into which he dived. No neurologic damage. Simple flexion injury with compression of the body of C5. (*D*) 13–50–40. Forty-eight year old male fell from a porch, landing on the back of his head. Transient paraplegia. Immediate roentgenograms not reproducible. Flexion injury with compression of the body of C6 and bilateral locking of facets. (*E*) 21–39–52. Twenty-six year old male who was thrown from a car that had skidded into an embankment. Paraplegia. Flexion injury with compression and unilateral locking of a facet, compression of the body of C7, spine fracture, and anterior chip. (*F*) 18–81–51. Twenty-nine year old male who was riding in the back seat of a car which overturned. He was thrown from the vehicle. Paraplegia. Hyperflexion injury demonstrating extreme comminution of the body of C7 and a "scooped out" defect.

vertebra above may undergo rotary dislocation.

As rotary dislocation of the vertebral body above occurs, the cord will be impinged upon by the posterior aspect of the vertebra below and the lamina above. The

neurologic damage therefore tends to be on the same side as the fracture or locking.

B. *Flexion with Compression.* If the direction of the force has a significant downward component, it will result in varying amounts of compression of the vertebral bodies where the force is resolved.

Roentgenographically, this type of injury presents with some amount of compression of the vertebral body with or without fractures in the posterior elements and with or without locking of the facets. This injury may also be unilateral. An anterior chip as described above may occur.

C. *Hyperflexion.* Hyperflexion injuries are caused by an acute flexion of the cervical spine followed by a bursting downward force. A common and easily understood sequence of events causing this injury is a particular fall. The initial forces are delivered to the back of a flexed head, breaking the posterior ligamentous structures (with or without fracture of the posterior elements), and stripping the anterior longitudinal ligament from the vertebra above the point where the forces are resolved. This acts to "cock up" the vertebra above the posterior ligamentous tear. The weight of the body then comes into play. The feet fall so that they come down on the toes, trapping the head under the chest. This results in a bursting force which either pushes the anterior inferior aspect of the vertebral body above into the substance of the vertebra below or collapses the vertebra above completely. This mechanism frequently results in neurologic deficit. The cord is damaged by being stretched across the posterior aspect of the end plates of the involved vertebrae during the acute flexion or by being injured by a posterior fragment of the vertebral body thrust into the canal.

Roentgenographically, this may present either of two pictures. The first is an almost complete collapse of a vertebral body with compression of its anterior and posterior height. The second may have normal alignment but will have multiple fractures with a dug-out impression in the posterior superior surface of the vertebral body below with normal anterior height. Fractures of the posterior elements of the vertebra are variable according to the breakage in the posterior ligamentous structures.

II. EXTENSION INJURIES

Extension injuries are caused by forces applied to the anterior skull or face. This type of force tends to drive the occiput back toward the dorsal spine thus stretching the anterior longitudinal ligament. If rupture in the anterior longitudinal ligament occurs and the force is sufficient to carry the vertebra above backwards, the vertebral body above the break may separate from the vertebral body below through the cartilaginous end plate leaving the disk intact, but stripping the posterior longitudinal ligament from the vertebra below. The spinal cord is carried backward with the vertebra above and may be contused against the leading edge of the lamina of the vertebra below. A chip from the anterior inferior surface of the vertebral body above or the anterior superior end plate of the vertebral body below may be pulled off according to the location of the break in the anterior ligament. Several months later the ligament may calcify at the point of breakage and an anterior fusion may occur (Fig. 3 and 4, *A–F*).

A. *Extension without Compression.* If the force is directed posteriorly and upward and is lifting in nature, only minimal bony injury may result.

Roentgenographically, the alignment varies. The vertebra usually settles back completely from the extended state, only rarely leaving a widened interspace anteriorly, with the two adjacent spinous processes closely approximated. There may be no roentgenographic signs of injury with complete settling of the vertebra above except for prevertebral soft tissue swelling or only an anterior chip.

B. *Extension with Compression.* If the principal lines of force are posterior and downward, they will produce a crushing of the posterior elements of the vertebra.

Roentgenographically, the alignment of

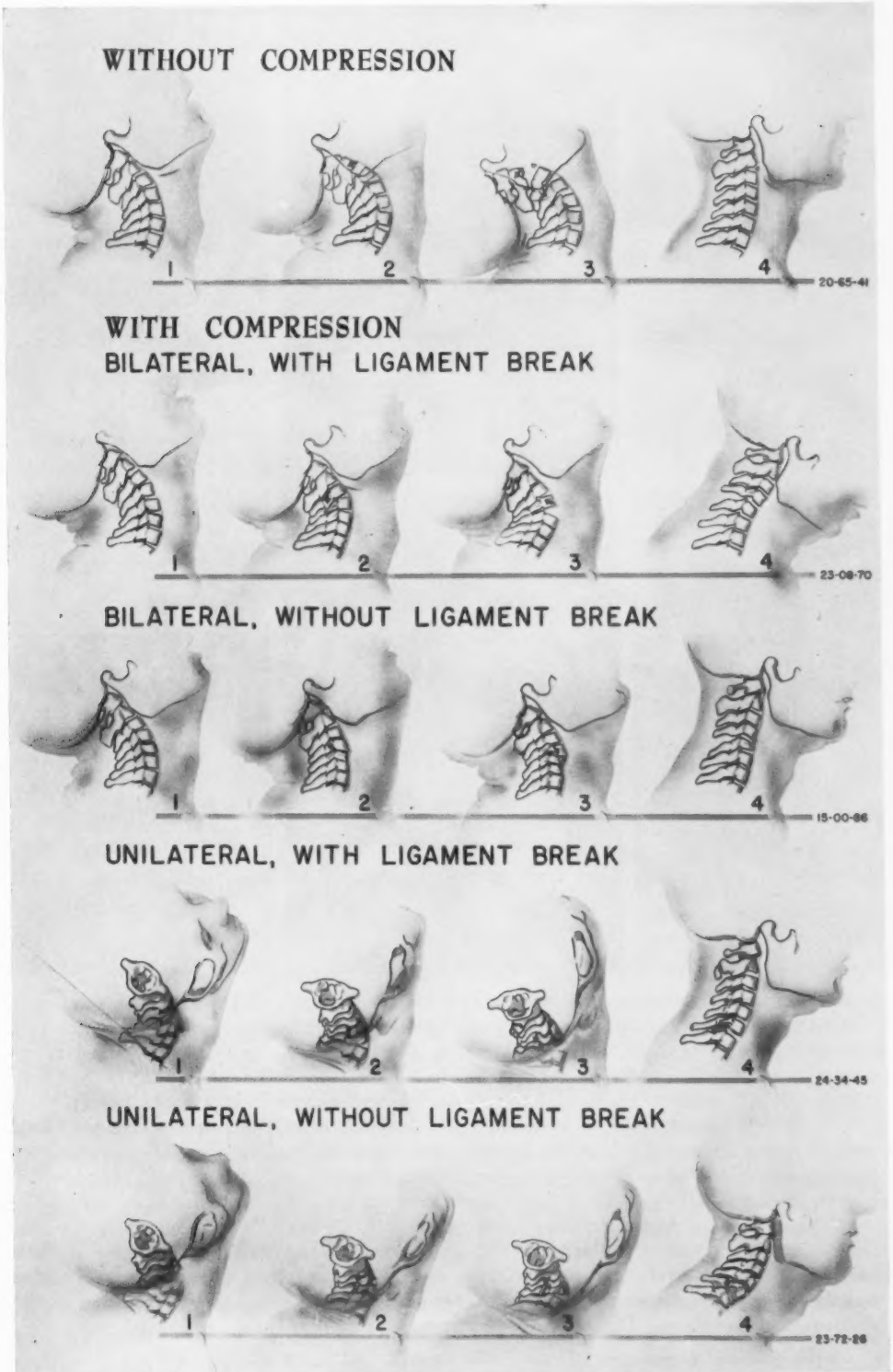

Fig. 3. *Extension Injuries.* Each line of drawings illustrates the dynamics of a single type of injury (1 through 3) and a typical end result (4).

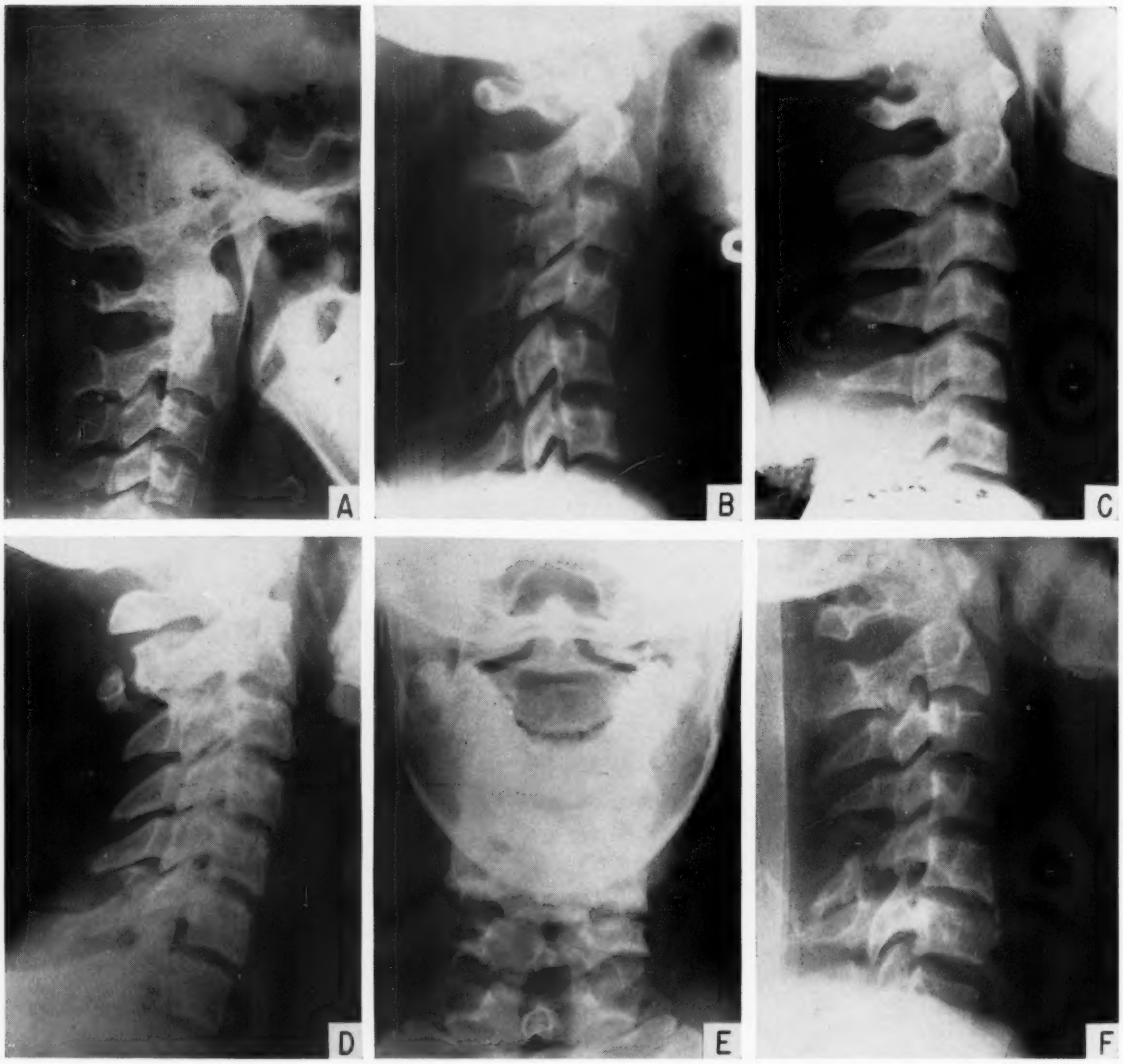

Fig. 4. The actual cases of the *extension injuries* illustrated in Figure 3. (*A*) 20-65-41. Thirty-five year old male epileptic with a history of falling while in epileptic seizure. Subsequently proved to have a left temporal astrocytoma. No neurologic findings. Extension injury without compression demonstrating only an anterior chip from C2 pulled away by the anterior longitudinal ligament. (*B*) 23-08-70. Sixteen year old boy who was injured in a diving accident. Tetraplegia. Bilateral extension injury with compression (hyperextension) illustrating the impacted inferior facets of C_4 and the small degree of forward subluxation commonly seen with this injury. (*C*) 15-00-86. Twenty year old male dived into a pool containing one foot of water. No neurologic signs. Hyperextension injury without ligament break illustrating the facet deformities and two anterior chips. (*D*) 24-34-45. Twenty-seven year old male who was driving an automobile which overturned. No neurologic signs. Unilateral hyperextension (rotary) injury demonstrating a unilateral facet deformity and a fracture of the lamina. (*E*) 24-34-45. An anteroposterior view of Case *D*, showing unilateral compression of the lateral mass of C6 on the right. (*F*) 23-72-26. Twenty-seven year old male passenger was thrown from an automobile after a collision. Paraplegia. Unilateral hyperextension injury without ligament tear, with unilateral facet deformity, fracture at the base of the spine and two anterior chips.

the vertebra about the break will depend on the type of injury and upon the handling of the patient after the injury. The inferior articulating facets of the vertebra above the point where the forces are resolved are impacted. On the lateral roentgenogram, the inferior facet tends to be horizontal and the lateral mass almost triangular in shape. (Fig. 6*F*). Fractures in the pedicles may result in dorsal displacement of the lateral

masses and spinous processes, which cause the unfractured facets to appear horizontal. Fractures in the spinous processes and lamina of the vertebra above the break, and other more distant vertebrae are common due to the direct contact of one spinous process upon another in the extreme extended state.

A break in the anterior longitudinal ligament may or may not occur after the separation of the verbebral body from the disk. According to the manner in which the forces resolve, the ligament may be severed and the interspace may open like a book; or the ligament may remain intact, and the vertebra above the separation may be pushed forward. The second mechanism may result in both a chip from the anterior body of the vertebra above and the one below the involved interspace.

C. *Unilateral Extension.* Rotary (unilateral) extension injuries are usually caused by blows to the lateral aspect of the face or temple with turning of the face away from the blow. (The original position of the head and angle of the blow may complicate the situation.) There is usually a downward or crushing component and the injury of the lateral mass will be an impaction or clean fracture of an inferior articulating facet on the side opposite the facial injury.

Roentgenographically, there may or may not be a gross abnormal curvature of the cervical spine in the anterior posterior projection. A constant finding will be a unilateral fracture of one of the inferior articulating facets which will be best seen in the lateral or oblique views. There also may be present a rotary dislocation of the vertebrae above the injury. Both unilateral extension and unilateral flexion injuries tend to cause unilateral neurologic damage; however, in the more severe cases the signs are bilateral. With severe unilateral extension forces, the anterior longitudinal ligament is usually severed on the side opposite the blow. As the vertebra above undergoes rotary dislocation, the posterior longitudinal ligament will carry the cord back and injure it upon the leading edge of the lamina of the vertebra below. Thus, in the example case, the bony and neurologic damage occurs on the side opposite the blow.

III. COMBINED INJURIES

Combined injuries, those resulting from flexion and extension forces of the cervical spine, are surprisingly rare. This is particularly interesting in view of the fact that a great number of cases result from automobile accidents where it would seem that the neck could undergo many different stresses. It is frequent that the cervical spine will be fractured in multiple locations; however, whether the fractures occur in the same, adjacent, or widely separated vertebrae, the same type force is usually causative. The case illustrated in Figure 5 and 6 *A* demonstrates a combined injury which occurred due to two separate forces acting in rapid sequence.

Two cases have been seen where it seemed logical that one single or continuing force resulted in a combined injury. In one, the injury was caused by a ten foot fall from a ladder. The patient struck the occiput with his head flexed, causing compression fractures of the bodies of C2 and C3. Then with his head relatively stationary, his body toppled backwards (with his heels striking the ground) causing a hyperextension injury at C6–C7. The injury resulted in paraplegia at the C7 level.

IV. BURSTING INJURIES

Bursting fractures are caused by blows directed downward on the vertex of the skull with the spine in neutral position and may result in compression of both the anterior and posterior elements of one or more of the cervical vertebrae. This type of injury can be converted into a flexion or extension type with only a minimal angulation of the injuring force and it is thus relatively rare as a pure entity (Fig. 5 and 6, *B* and *C*). The most common pure example of this type of injury is "Jefferson's fracture,"[3] which consists of the bursting of the ring of C1 by the occipital condyles being driven downward.

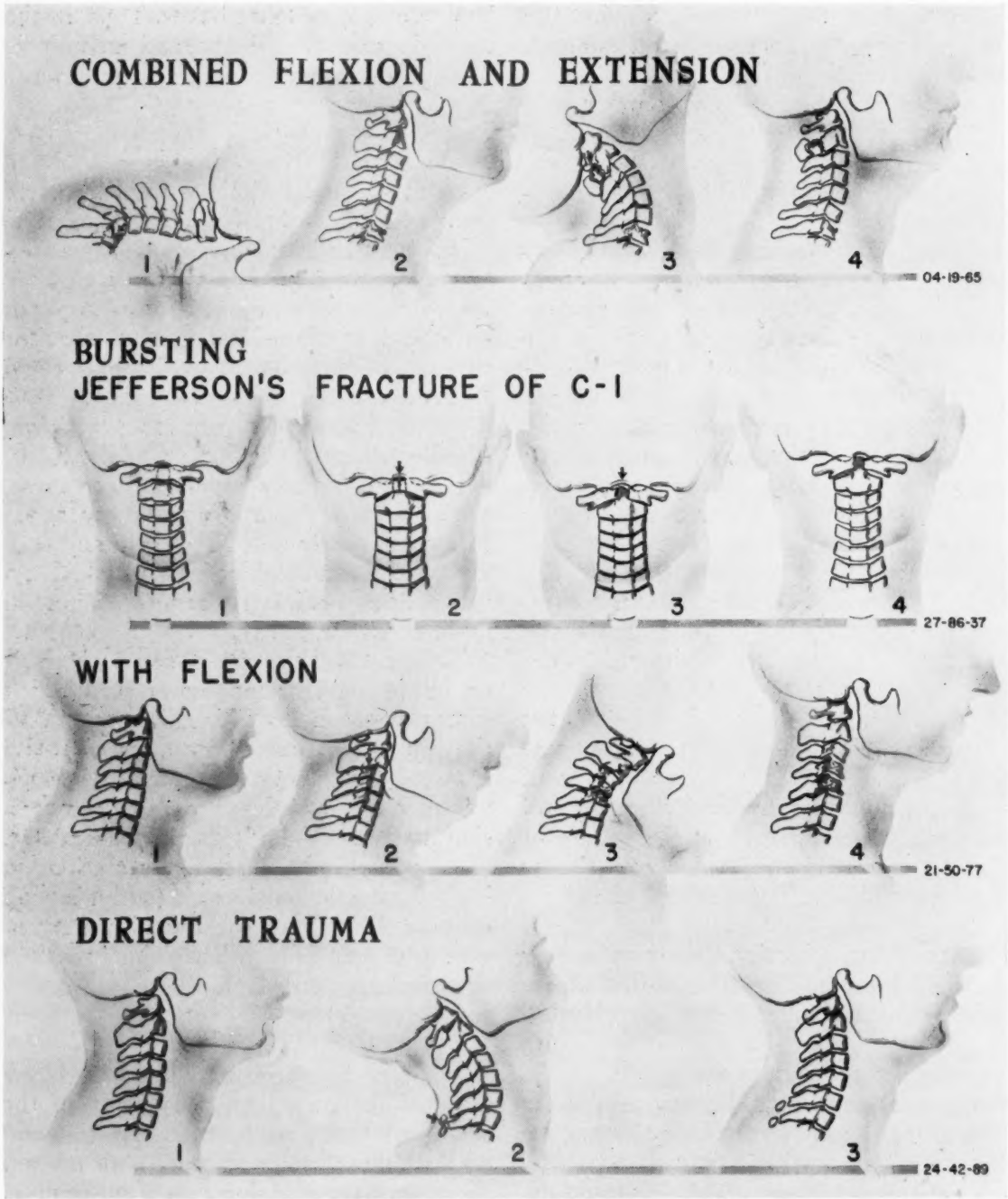

Fig. 5. *Combined Injuries and Miscellaneous.*

V. LATERAL FLEXION

The lateral flexion injuries are caused by a force delivered directly to the lateral aspect of the cervical spine and skull. They result in wedging of the lateral aspect of a vertebral body and its associated lateral mass. A case of this type did not occur in our series. Those reported in the literature occurred almost solely in the pediatric age group.

VI. DIRECT TRAUMA

A direct blow to the neck over a spinous process can result in a fracture of this struc-

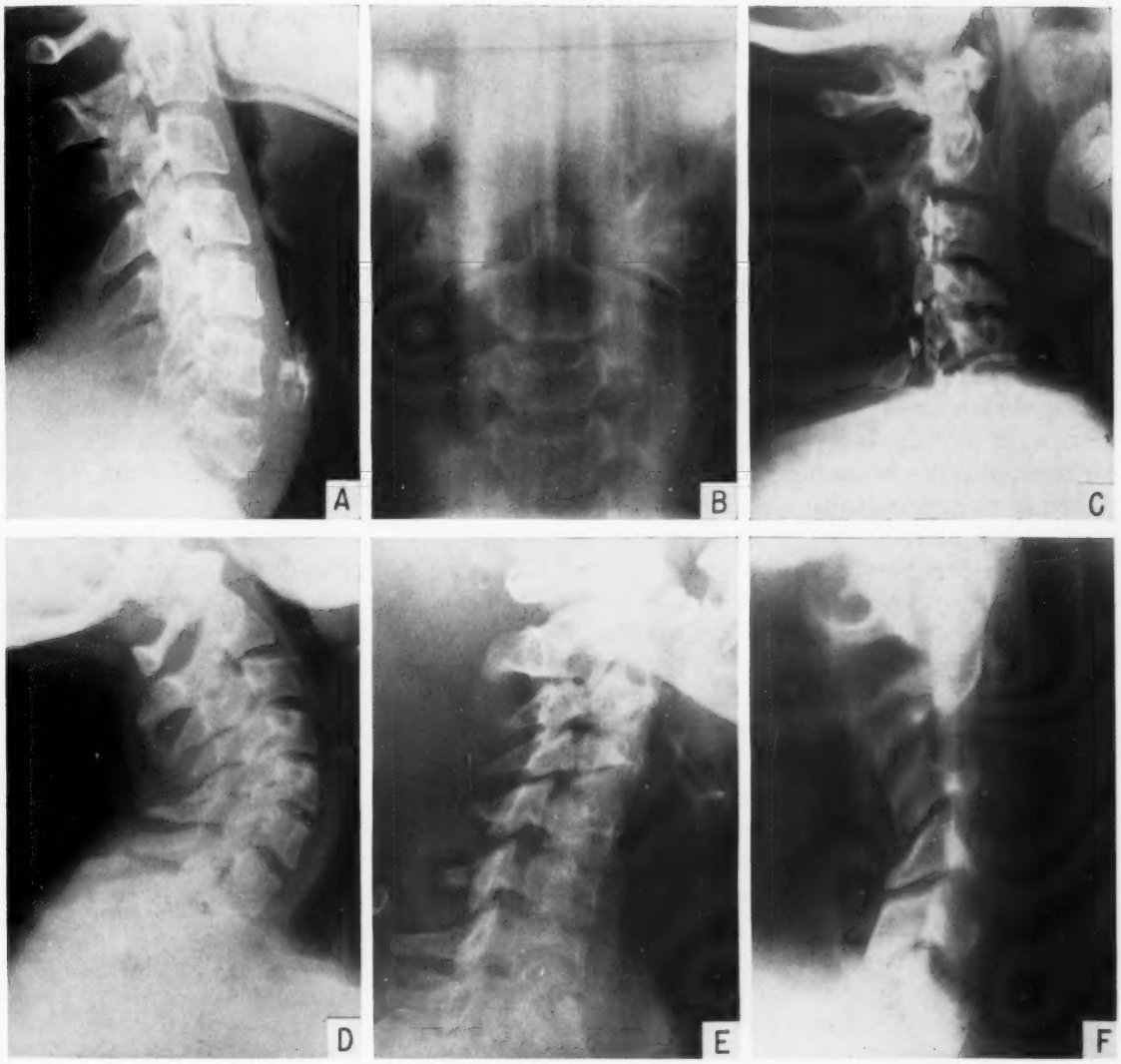

Fig. 6. The actual cases illustrated in Figure 5. (*A*) 04-19-65. Twenty-eight year old male who was riding in the front of a speed boat which ran under a cable. He was struck at the base of the neck, flipped into the air, and fell on his face in the back of the boat. Combined hyperflexion injury of C_7 and hyperextension injury of the lamina and pedicles of C_2. (*B*) 27-86-37. Thirty-five year old male who was hit by a falling tree. Hypesthesia of the posterior scalp and temporarily of the right hand. He was struck on the vertex of the skull. Bursting injury of C_1 (Jefferson's fracture). Laminagram shows lateral displacement of the lateral masses. (*C*) 21-50-77. Sixteen year old male sustained injury from diving into a shallow creek. Tetraplegia. Bursting injury with some flexion of C_4. Diminished anterior and posterior height of C_4 vertebra. (*D*) 24-42-89. Twenty-three year old female who was struck over the lower neck with a baseball bat. Fractures of the spinous processes of C_7 and T_1. (*E*) An oblique roentgenogram demonstrating the typical deformity of the inferior facet of C_4, occurring secondary to an extension injury with compression. (*F*) Laminagram of Case *E* performed in the oblique projection.

ture. In our experience this fracture usually occurs in the lower cervical spine (Fig. 5 and 6 *D*).

Penetrating missiles may also produce fractures of the vertebrae as well as neurologic damage.

TECHNIQUE OF EXAMINATION OF THE CERVICAL SPINE

The most important single aim in the examination of a patient with a neck injury is to prevent further damage. Experience has taught us that this can be best accomp-

lished as a cooperative effort between the orthopedic, neurosurgical and radiologic services.

Patients suspected of having cervical spine injuries are placed on a stretcher with 5 to 7 pounds of halter traction in the emergency room prior to roentgen examination. If the patient has an obvious cervical spine injury with neurologic damage, 10 to 20 pounds of skull traction is applied by means of Crutchfield tongs prior to leaving the emergency room. The direction of traction is always kept *straight* (in line with the vertical axis of the body) until satisfactory roentgenograms have demonstrated the type and location of injury, and the mechanism of injury is ascertained.

The first roentgenogram obtained in our department on such patients is a lateral horizontal beam view of the cervical spine, which is checked by a radiologist for gross fractures. If there is no contraindication from the initial roentgenogram, then Bucky open-mouth roentgenograms of the odontoid and full anteroposterior roentgenograms of the cervical spine are obtained. Occasionally, oblique, stereoscopic, and flexion and extension roentgenograms in the lateral and oblique projections are required. Barring complicating factors, the patient remains in the radiologic department until satisfactory roentgenograms have been obtained.

The lateral view of the cervical spine has proved to be of the greatest diagnostic value in our initial examination. This view can be obtained in almost all cases. In a well-penetrated roentgenogram, one is able to visualize the bodies and posterior structures.

The oblique view perhaps demonstrates to best advantage the pedicles, lateral masses and laminae (Fig. 5 and 6, *E* and *F*). However, satisfactory oblique roentgenograms can rarely be obtained initially with a serious, acute cervical spine injury.

The anteroposterior view is most useful in the rotary (unilateral) injuries, where frequently a difference in the vertical dimensions of the lateral masses can be shown. It also discloses to best advantage vertical fractures in the vertebral bodies. Slight caudal angulation of the beam will help visualize the height of the lateral masses. Cephalad angulation will aid in demonstrating the vertebral bodies. The exact angles for these two variations of the anteroposterior view can be judged in each case by the horizontal beam lateral roentgenogram.

The open-mouth roentgenogram of the odontoid serves to the greatest advantage in determining the integrity of the odontoid process of the second cervical vertebra and in judging the heights of the lateral masses of the first cervical vertebra.

Flexion and extension views are particularly useful in demonstrating tears in the posterior ligamentous structures. Once they are torn, ligaments rarely heal and the presence of such tears may be ascertained long after the initial injury. A special note should be made that extension views of the cervical spine are extremely dangerous in the acute case when there is a serious suspicion of breakage of the anterior longitudinal ligament. If the head is tilted back far enough and the ligament is not intact, then a posterior dislocation may recur causing or worsening neurologic damage.

Roentgen Signs in the Differentiation of the Types of Cervical Spine Injury

1. Anterior wedging of a vertebral body, with or without a vertical fracture, signifies that the force involved had a significant flexing force with some degree of compression.

2. Anterior and posterior compression of a vertebral body or extreme comminution with depression of its posterior superior surface implies hyperflexion.

3. An anterior chip fracture from a vertebral body may arise from either a flexion or extension injury. The majority of those arising from the anterior inferior margin were felt to be secondary to extension injuries while the majority of chips from the anterior superior margin were due to flexion

injuries. A chip from the anterior inferior margin of a vertebral body is thought to be more likely to occur with a tear in the anterior longitudinal ligament.

4. Unilateral and bilateral complete dislocation with locking of facets is due to flexion forces.

5. Impaction of the inferior articulating facets of a vertebra, or fracture of the pedicles with the facets assuming a "horizontal" position, always indicates an extension injury.

6. Impaction or chip fracture of a superior facet usually implies a unilateral flexion force without significant compression.

7. Loss of vertical height of one lateral mass of a vertebra (as seen in the anteroposterior view) always indicates a significant unilateral component to the injuring force.

8. Fractures of the spinous process may be due to direct trauma, flexion or extension. Cephalad displacement of a fractured spinous process implies extension.

TECHNIQUE OF ANALYSIS

An analysis of 159 cases was made correlating the clinical with the roentgenographic findings. All cases were investigated as to the history of how the injury was inflicted, the associated injuries, the initial and subsequent roentgenographic studies, the operative findings (if operation was performed), and, where possible, the end clinical result. Myelographies were performed in 29 cases. Operative procedures on the cervical spine injury were performed in 31 per cent.

RESULTS

The cases of cervical spine injuries analyzed by the above proposed classification

TABLE I

CLASSIFICATION OF THE SERIES ACCORDING TO THE TYPE OF INJURY AND THE NEUROLOGIC FINDINGS ON ADMISSION TO THE NORTH CAROLINA BAPTIST HOSPITAL

	Negative Neurologic Findings	Paraplegia	Tetraplegia	Unilateral Signs	Total
A. Injuries to C1 and C2					
Flexion	11		9		20
Extension	19		1	2	22
Bursting (Jefferson's fracture)	1				1
B. Injuries below C2					
Flexion without compression					
bilateral	6	18	9		33
unilateral	5	1		2	8
Flexion with compression					
simple	3				3
bilateral	2				2
unilateral	2			1	3
Hyperflexion	1	4			5
Extension without compression	9	3			12
Extension with compression					
bilateral	12	7	1		20
unilateral	7	2	4	4	17
Combined flexion and extension		2	1		3
Direct trauma (including gunshot)	4	3	2	1	10
	82	40	27	10	159

Total injuries of C1 and C2: 43. Total injuries below C2: 116.
Total flexion injuries: 73.
Total extension injuries: 71.

are shown in Table 1. The series contains 23 females and 136 males. The mechanism by which the injuries were sustained were: 87 automobile accidents; 31 falls; 13 diving accidents; 7 injuries secondary to gunshot wounds; and 21 other. The average age of the victims was thirty-one years, with a range from eight months to eighty-one years.

DISCUSSION

The extent of bony injury does not necessarily reflect the severity of the neurologic damage in many cases. This was well illustrated by Taylor and Blackwood,[6] who autopsied 2 cases of cervical spine injury resulting in death, in which no roentgenographic evidence of bony injury was present. To correlate the clinical symptomatology of some patients with the roentgenographic findings requires the proposal of a mechanism of injury. Determining from the roentgenograms alone the mechanism of injury may be a difficult or an impossible task in a single case. We have found that the history of how the injury occurred and the knowledge of the associated bony and soft tissue trauma is of great value in leading to the final diagnosis in less obvious cases.

The differentiation of the mechanism of injury is of the utmost importance in the proper handling of the patient as well as in instituting therapy. If an extension injury is mistaken for a flexion injury, and the neck is manipulated with hyperextension to obtain extension roentgenograms or to correct the deformity, the most dire consequences may result.

It is common for flexion and extension injuries to have a unilateral or rotary component. In some cases it is difficult to decide from the roentgenograms alone which is of greater importance in the clinical picture. If present, unilateral neurologic signs are usually a very significant indicator.

The further subdivision of flexion and extension injuries into those with and without compression was undertaken in order to substantiate the clinical impressions that: (1) complete dislocation of the facets with locking (and neurologic damage) was less common in flexion injuries with a significant compression of the vertebral body; and (2) neurologic deficit is more common with extension injuries when compression of the posterior elements is present (at least in this type of "alive on admission" series).

CONCLUSIONS

A classification of fractures of the cervical spine according to their mechanism of injury is proposed. A series of 159 cases is analyzed. A technique for the handling of such patients is presented.

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LAMINAGRAPHY IN THE FOLLOW-UP OF FRACTURES OF THE ODONTOID PROCESS*

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MONTGOMERY, ALABAMA

TEN cases of fracture of the odontoid process have been treated and followed at this hospital (Table 1). Interest in this fracture was stimulated by the profound importance of determining the end result—bony union or nonunion.

ANATOMY

The odontoid process, tooth-like in shape, projects upwards from the superior surface of the body of the second cervical vertebra or axis and forms a pivot upon which the first cervical vertebra, carrying the head, rotates. The stability of the head on the neck depends on the integrity of this pivot. The odontoid is located directly anterior to the spinal canal. When fractured, it may slip anteriorly or posteriorly with the atlas, causing encroachment on the spinal canal[5] and possible spinal cord injury. Fracture of the odontoid process generally occurs at its base and may extend into the upper anterior portion of the body of the axis.

LAMINAGRAPHIC EXAMINATION

Routine anteroposterior and lateral roentgenograms of the cervical spine and open mouth roentgenograms of the odontoid process have often been unsatisfactory in the diagnosis and follow-up of this fracture. The fracture line is frequently difficult to visualize in the open mouth roentgenogram because of its obliquity from behind forward, and because of its location in cancellous bone. In the lateral roentgenogram, the fracture may be obscured by the superimposed structures of the atlas. The fracture is frequently suspected from a change in the normal alignment of the odontoid process or from a slight tilt in one direction or another. Laminagraphy may be required to confirm the presence of fracture, and it is the method of choice in follow-up examinations.

FOLLOW-UP STUDIES

The end results of fracture of the odontoid process are either firm bony union which may occur in from four to nine months,[1] firm fibrous union, or loose fibrous pseudarthrosis. The latter two are actually nonunion, since there is no bone joining the fragments. However, in firm fibrous union the fragments are held close together by the fibrous tissue, and there is little if any movement between them. The fragments may at times be so closely approximated as to simulate bony union on routine roentgenograms, but the space between them can be seen in laminagraphy (Fig. 1, *A*, *B* and *C*; 2; and 3 *A* and *B*). In loose fibrous union, there is considerable absorption of bone about the fracture line, and the odontoid fragment is widely separated from the body of the axis. It may show a variable degree of atrophy and osteoporosis and it may be adherent to the anterior arch of the atlas.[2] This condition is readily recognized on routine roentgenograms, and laminagraphy is not essential, though providing additional detail (Fig. 4).

In our group of cases, 4 healed by bony union (Fig. 5, *A*, *B* and *C*), 3 by firm fibrous union, 2 by loose fibrous pseudarthrosis, and 1 was not followed long enough to determine the result. Previously reported groups of cases of fractures of the odontoid process showed considerable variation in the per cent that united with bone,[1,2] and the differentiation between bony union and firm fibrous union was not always made.[1]

With bony union, the patient may re-

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TABLE I

Age and Sex	Type of Injury	Associated Injuries	Roentgen Ray Findings	Neurologic Complications	Treatment	Results
W.P. 41 Male	Automobile accident	None	Odontoid displaced slightly anteriorly and tilted to the right	None	Cervical brace	Bony union in four months and one week. No change in position of odontoid
H.S. 48 Male	Automobile accident	None	Fracture of base of odontoid, extending into anterior portion of body of C_2. Slight anterior and downward displacement of fragment	None	Extension cervical brace at first. Crutchfield tongs inserted at two weeks for four weeks on account of anterior slipping of odontoid. Cervical brace applied for six months after anterior displacement was corrected	Firm bony union—followed three years
E.D. 34 Male	Automobile accident two weeks before admission	None	Odontoid displaced slightly anteriorly and tilted slightly to the left	None	Traction for two weeks. Later, cervical extension splint for six months	Firm bony union in nine months. Position unchanged
R.H. 33 Male	Automobile accident five weeks before admission	Slight compression fracture of sixth dorsal vertebral body	Odontoid displaced anteriorly very slightly	None	Cervical brace for first five weeks. Schanz type of cervical collar for four weeks	Partial bony union in two months. Seen one year and eleven months later. No symptoms referable to the neck, but not roentgenographed
L.V. 32 Male	Automobile accident two weeks prior to admission	Fracture of posterior arch of atlas	Odontoid displaced slightly posteriorly	None	Bed rest with quilted collar support. Later, cervical brace worn for six months	Firm fibrous union demonstrated two years, eleven months later
S.G. 32 Male	Automobile accident	Cerebral concussion	Odontoid displaced slightly posteriorly and tilted slightly to the right and posteriorly	None	Cervical brace after two weeks, for six months. Could not be treated earlier on account of cerebral concussion	Firm fibrous union demonstrated four years, nine months later
J.E.F. 33 Male	Automobile accident ten days prior to admission	Fracture right femur and left scapula	Odontoid displaced and tilted anteriorly, and tilted slightly to the left	None	Crutchfield tongs traction for six weeks. Later, modified Schanz cervical collar for four weeks	Firm fibrous and bony union demonstrated two years, four months later. Anterior displacement partially corrected
L.D. 64 Male	Fall from porch steps six days before admission	None	Odontoid process displaced slightly posteriorly and to the left; tilted slightly to the left	None	Neck halter traction for one week. Later, cervical brace with dorsal spine extension support for six months	Loose fibrous pseudarthrosis. Followed three years and two months. No symptoms referable to the neck
L.S.S. 21 Male	Automobile accident nine days before admission	None	Odontoid process displaced slightly anteriorly and to the right	Early numbness of left hand and weakness of left hand grip. Inability to void. All symptoms cleared up after traction started, and did not recur	Refused treatment for first two weeks. Neck halter traction for one week. Cervical brace with extended dorsal spine support worn in daytime and Schanz type of collar at night for six months	Loose fibrous pseudarthrosis. Followed three years and three months
M.H. 35 Male	Fall at home when foot became entangled in trousers leg	None	Odontoid process displaced slightly anteriorly	Slight weakness of grip of both hands. No sensory loss or reflex changes. No bladder or bowel symptoms. Cleared up in a few days	Cervical brace for sixteen weeks. Did not return for further care	Followed five months with no signs of union. No further follow-up

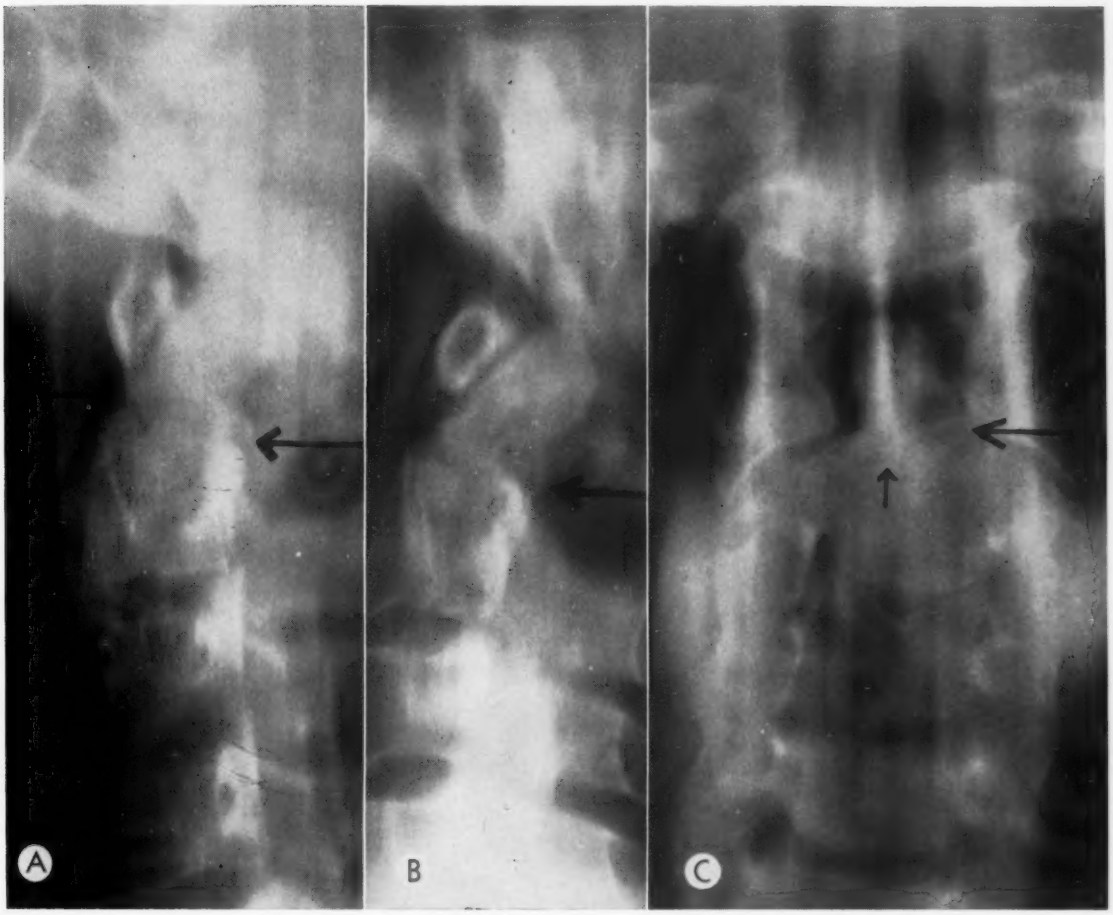

Fig. 1. S.G. Firm fibrous union. Laminagrams made four years, nine months after injury show only slight movement of the odontoid process with head in (*A*) flexion and (*B*) extension. (*C*) Anteroposterior laminagram shows unobliterated fracture line.

sume normal activity without danger of late neurologic complications. Firm fibrous union is compatible with safety, as there is no tendency for the odontoid to slip with the passing of time,[2] unless additional trauma is incurred. With loose fibrous union, a sudden jerk of the head or violent activity may cause luxation of the odontoid and compression of the spinal cord. Osgood and Lund[6] state that, in some of their patients who had been treated for fracture of the odontoid process, a later trivial accident precipitated fatal paralysis. The type of healing in these cases is not mentioned but it is presumed not to have been with bone. Delayed neurologic complications are at times due to other causes than displacement of the odontoid, such as arachnoiditis, myelitis, osteomyelitis, or excessive callus formation.[2,7,8]

The normal pivotal action of the odontoid takes place between it and a ring formed by the anterior arch of the atlas in front and the transverse ligament of the atlas behind. The first cervical vertebra, which turns about the odontoid, rotates on the axis at the atlanto-axial joints. With an ununited fracture of the odontoid, a variable degree of instability develops at the atlanto-axial joints, permitting some side to side movement and anteroposterior movement when the head is moved to one side or the other or forwards and backwards.[1] The 2 patients in our series with loose fibrous pseudarthrosis of the odontoid showed considerably more than normal

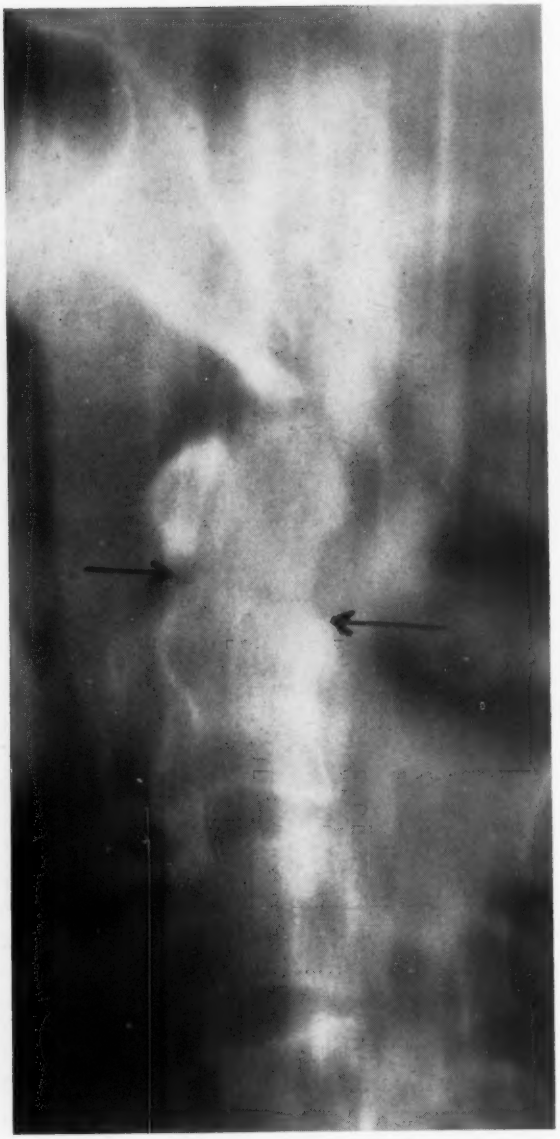

Fig. 2. L. V. Firm fibrous union. Laminagram made two years, eleven months after injury.

motion at the atlanto-axial joints in unrestricted flexion and extension of the head and also lateral flexion three years after the injury (Fig. 6, *A* and *B;* and 7, *A* and *B*). However, this was not sufficient to produce any symptoms referable to the neck or signs of spinal cord compression. The laxity of the articular capsules of the alanto-axial joints permits this excessive motion, but some stability is nevertheless obtained from the strong ligaments joining the upper cervical vertebrae and skull preventing a range of motion which would cause spinal cord compression. Grogono[4] divided the transverse and accessory ligaments of a fresh necropsy specimen from an elderly subject and found that considerable force was required to cause displacement of the atlas. Fritsche[3] sawed through the base of the odontoid on a cadaver but found that luxation between the atlas and epistropheus could be produced only by cutting through the ligaments of the upper cervical region. These experiments demonstrate the strength and support provided by the ligaments of the upper cervical spine, unless they are also injured.

Rotation of the neck in both of these patients also produced no untoward signs or symptoms. Roentgenograms made in a rotated position of the head showed apparently normal rotation at the atlanto-axial joints (Fig. 4, 8 and 9). It was as though the odontoid was still functioning as a pivot, even though detached. A plausible explanation is that the muscles of the neck are so well co-ordinated from long experience that they continue to pivot the head and atlas about the proper axis of rotation.

SUMMARY

1. The odontoid process is important because the stability of the head depends on this process, and, in case of fracture, its sublaxation or dislocation may narrow the spinal canal and damage the spinal cord.

2. The fracture may heal by bony union, firm fibrous union, or loose fibrous pseudarthrosis. Fibrous union results in increased susceptibility to subsequent trauma. With firm fibrous union the atlanto-axial joints are relatively stable, but there is moderate instability with loose fibrous union. Movement and rotation of the upper cervical spine are nevertheless accomplished without symptoms or signs referable to the neck or spinal cord.

3. Routine roentgenography will show

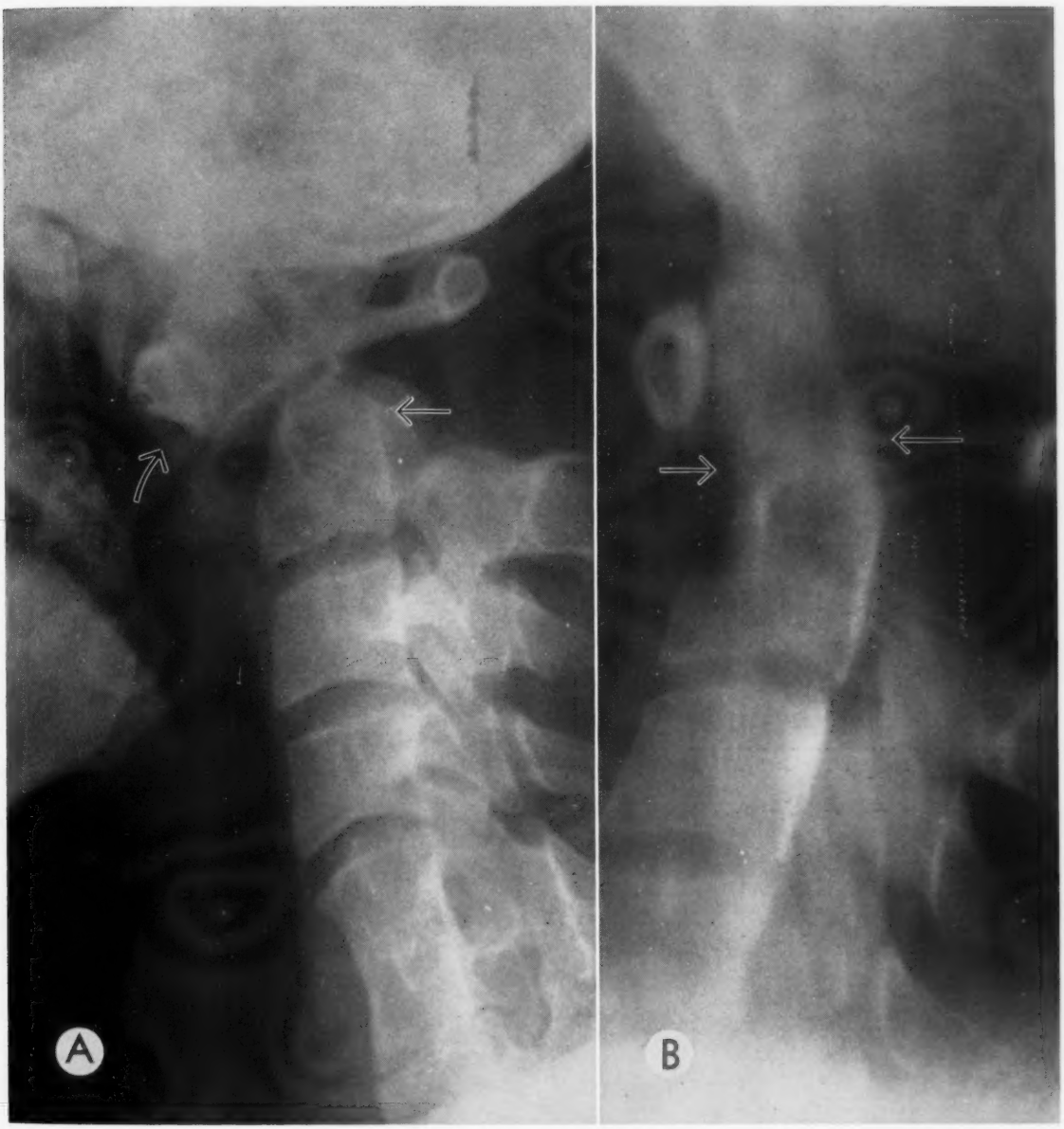

Fig. 3. J.E.F. Firm fibrous and bony union two years, four months after fracture. (*A*) Initial lateral roentgenogram showing fracture of the odontoid with anterior dislocation. (*B*) Lateral laminagram showing thin interrupted radiolucent line at the base of the odontoid, believed to represent healing by bone interspersed with areas of union by fibrous tissue.

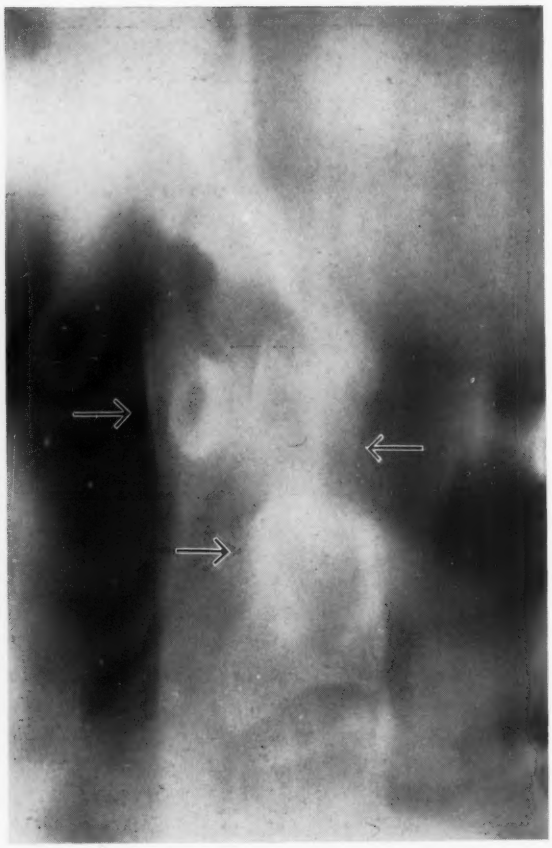

Fig. 6. L.D. Sixty-four year old white male with loose fibrous pseudarthrosis of the odontoid three years, two months after the injury. The odontoid fragment appears to be adherent to the anterior arch of the atlas. Laminagrams made with head in (*A*) flexion and (*B*) extension show anterior and posterior displacement of the odontoid fragment and the atlas.

Fig. 4. L.S.S. Loose fibrous pseudarthrosis of the odontoid in a twenty-one year old white male. Laminagram made three years, three months after injury with head rotated. The odontoid fragment is osteoporotic and atrophied. It appears to be displaced upwards.

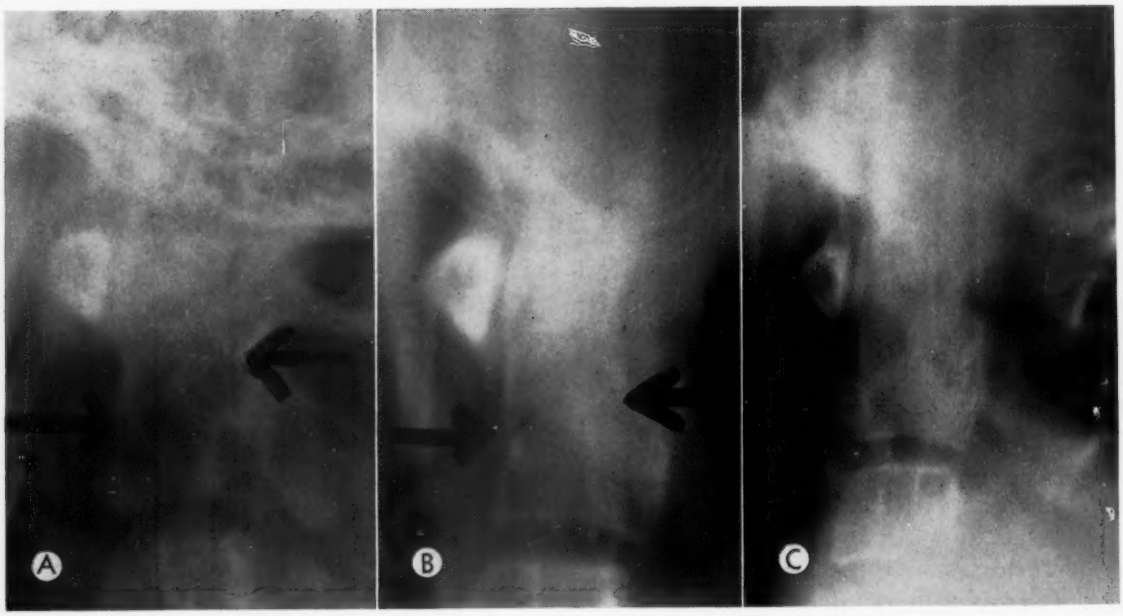

Fig. 5. W.P. Firm bony union of the odontoid four months, one week after fracture. (*A*) Routine lateral view of the neck shows oblique fracture of the base of the odontoid. (*B*) Lateral laminagram two months later shows blurring of the margins of the fracture. (*C*) Lateral laminagram two months later shows bony union.

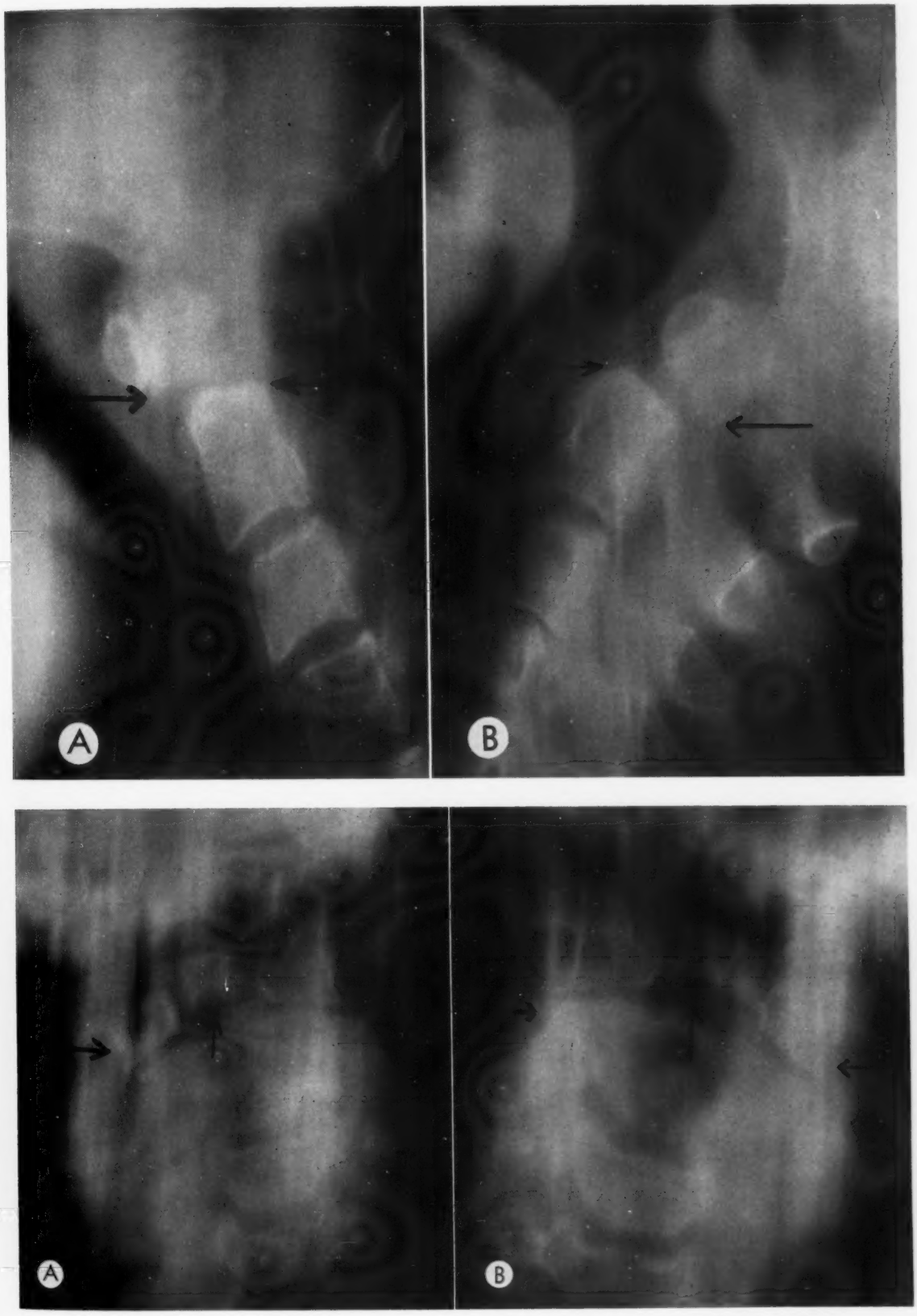

FIG. 7. L.S.S. Twenty-one year old white male with loose fibrous pseudarthrosis of the odontoid three years, three months after injury. Laminagrams made with the head in (*A*) right lateral flexion and (*B*) left lateral flexion show lateral displacement of the odontoid fragment and the atlas.

Fig. 8. L.S.S. Routine lateral view of the neck with the head rotated. The lateral masses of the atlas are located anterior and posterior to the detached odontoid fragment which is in its normal position.

a loose fibrous pseudarthrosis, but laminagraphy may be required to differentiate bony union from firm fibrous union.

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Fig. 9. L.D. Lateral laminagram with the head rotated shows the odontoid fragment in normal position and one of the lateral masses of the atlas posterior to it.

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CONGENITAL ABSENCE OF THE ODONTOID PROCESS*

REPORT OF A CASE

By J. GEORGE TEPLICK, M.D., STANLEY STEINBERG, M.D., *and* BERNARD P. ADELMAN, M.D.
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RARE indeed is congenital absence of the odontoid process of the second cervical body (axis). This anomaly was originally described by Roberts[8] in 1933. A search of the subsequent medical literature discloses only 15 reported cases.[1,3–12]

The odontoid process is formed by an upward extension of the cartilaginous mass of the body of the second cervical vertebra. Two ossification centers appear in the base of this cartilaginous process around the sixth month of fetal life. Before birth, these centers fuse to form a conical bilobed mass with a deep cleft above, which is filled with a wedge-shaped cartilage. The apex has a separate center, appearing in the second year of life and fusing with the other center about the twelfth year. The base of the odontoid is separated from the body by a cartilaginous disk, which ossifies at its circumference, but remains cartilaginous in its center until advanced age.[12]

Mere failure of center ossification results in an absent shadow of the dens on the roentgenogram. The failure of development of the cartilaginous odontoid process allows an abnormally great range of motion of the atlanto-axial bodies. Strong flexion, extension and lateral bending of the neck produce apparent dislocation of the atlas on the axis.

REPORT OF A CASE

R.R., a forty-two year old white male, had been injured in an automobile accident a few months prior to the roentgenographic examination. Although his clinical complaints were quite minimal, roentgenograms of the left elbow, left forearm, left wrist, left shoulder and the cervical spine were ordered and made for medico-legal purposes. The left elbow was deformed, as a result of an injury many years previously.

All roentgenograms of the extremities were normal, except those of the elbow, where evidence of an old fracture with deformity and soft tissue calcification was found.

The roentgenograms of the cervical spine (Fig. 2, *A* and *B*) showed absence of the dens shadow in the lateral view, with an unusually clear visualization of the lateral mass. In the anteroposterior view (Fig. 1), the findings were most striking. The dens was completely absent; the smooth margin of the axis, from which the dens normally projects upward, emphasized the congenital nature of the abnormality. Lateral bending led to roentgen evidence of subluxation of the atlas to the bent side. Flexion of the

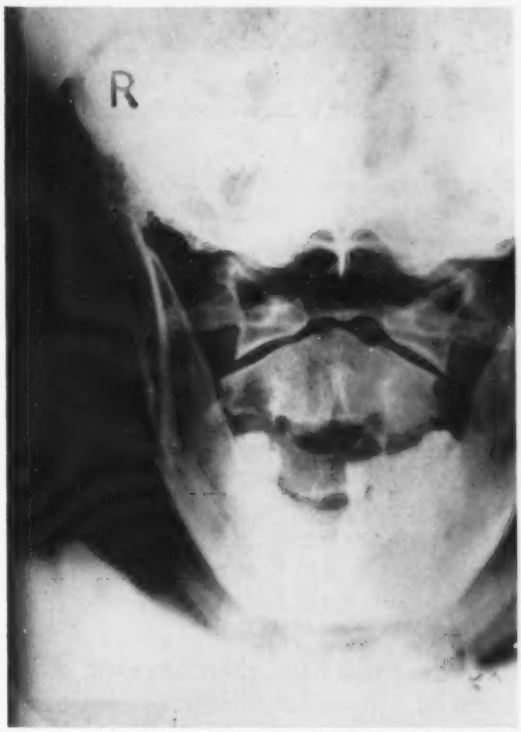

FIG. 1. The absence of the odontoid process in the anteroposterior view is self-evident. Note the smooth surface of the axis from which normally the odontoid projects. The patient's head is tilted slightly to his left, with obvious subluxation of the atlas on the axis.

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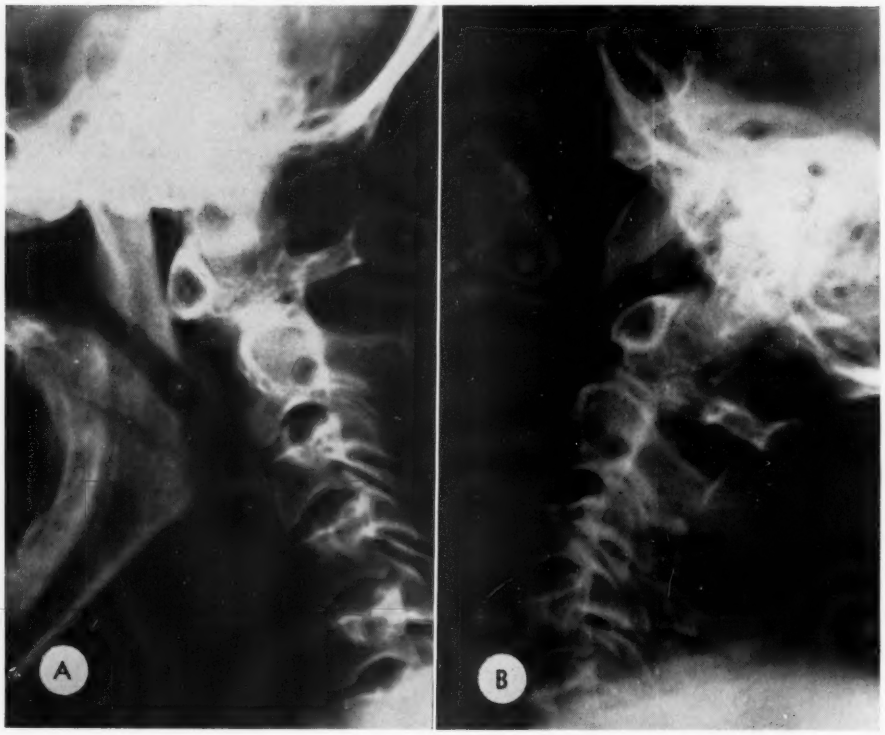

Fig. 2. (*A* and *B*) Lateral views in flexion and extension. Note the striking absence of the odontoid process. On flexion there is forward subluxation and on extension posterior subluxation of the atlas.

neck produced considerable forward subluxation of the atlas on the axis. None of the extreme motions elicited any discomfort from the patient.

Neurologic examination at a later date proved to be completely negative. The neurologist could find no evidence of cord pressure even with extremes of neck motion.

Our attempt to get some information on possible genetic transmission of this anomaly was unsuccessful. Our patient's parents were deceased; of 2 siblings, one was overseas and the other (sister) was embroiled in a family quarrel and was entirely uncooperative.

DISCUSSION

Including this case, there are only 16 instances of congenital absence of the odontoid process reported in the literature. Some interesting data can be gleaned from these. Only 4 were females. The abnormality was discovered at varying ages ranging from eight to fifty-six years. A history of trauma preceded the roentgen studies of the cervical area in all but 6 patients. Subluxation of the atlas on the axis was observed roentgenographically in 12 of the 16. We would hazard a guess that were roentgenograms made in extreme lateral and forward flexion, the subluxation would have been demonstrable in the other 4 patients. With a completely absent odontoid process, there is no anatomic mechanical bar to subluxation on positional changes of full range.

In only 5 patients were neurologic changes uncovered. Distinct evidence of cord compression after neck trauma was found in one patient.[3] This was relieved by traction. In another patient, [10] severe adhesive arachnoiditis was found in the atlanto-axial area, and he was partially relieved by decompressive laminectomy.

In 3 patients without neurologic findings, prophylactic operative fusion of the atlas with the axis was performed.[4,5,12] Ivie[3] recommends this operative procedure for all cases. Although the marked subluxation from extremes of neck position in most of the cases is not attended with cord com-

pression or injury, traumatic hyperextension or hyperflexion could theoretically produce serious results on the cord. It would seem prudent to warn any "adentulous" individual of this danger.

Indulging in fanciful speculation, we ponder the fate of an "adentulous" individual undergoing punitive hanging. Death from hanging is due to acute cord compression when the severe neck snap fractures the dens, acutely dislocating the atlas. Legends tell of some individuals who could not be hanged successfully. Were they "blessed" with congenital absence of the odontoid process?

CONCLUSIONS

1. A case of congenital absence of the odontoid process of the axis is reported, the sixteenth in the medical literature.

2. Although there were no neurologic signs or symptoms, extreme subluxation of the atlas on the axis was noted roentgenologically with extremes of neck position.

3. Individuals with this anomaly might be prone to cord injury after trauma to the neck. Whether this danger should be met by fusion operation or by mere caution to avoid neck trauma can hardly be settled in view of the paucity of reported cases.

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AN UNUSUAL VARIANT OF THE CERVICAL SPINE

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ON TWO recent examinations of the cervical spine, a bony prominence, squarish in shape and projecting anterior to the body of the sixth cervical vertebra has been observed on the lateral roentgenogram. The identity of this prominence is not generally known. Since accuracy of diagnosis in this region is important both from the clinical and medico-legal standpoints, the absolute identification of all roentgenographically visible structures is imperative. The purpose of this presentation is to clarify this diagnostic point.

ILLUSTRATIVE CASES

The first patient was a young man referred for examination following an injury to his neck in a swimming pool accident. No evidence of fracture, dislocation, or subluxation was seen. The examination did show evidence of muscle spasm and soft tissue injury (Fig. 1). The second patient had a past history of having received electroshock therapy and a recent onset of neck pain. This was aggravated by motion. In this patient, also, the unusual bony prominence was seen in the lateral projection of the cervical spine. No other abnormal findings were noted in the roentgenographic study of the cervical spine (Fig. 2).

In neither case was it possible to delineate the posterior aspect of the bony prominence from the body of the vertebra. It could not be definitely identified on either the anteroposterior or oblique roentgenograms of the spine, although lengthening of the transverse processes toward the distal end of the cervical spine was noted.

In the lateral view, with the spine in flexion and extension, the density maintained its relationship to the body of the sixth cervical vertebra (Fig. 3, *A* and *B*).

DISCUSSION

A study of the bones of the cervical spine

FIG. 1. Case I. Lateral view of the cervical spine showing the bony projection anterior to the body of the sixth cervical vertebra.

FIG. 2. Case II. Lateral view showing the unusual bony prominence anterior to the body of the sixth cervical vertebra.

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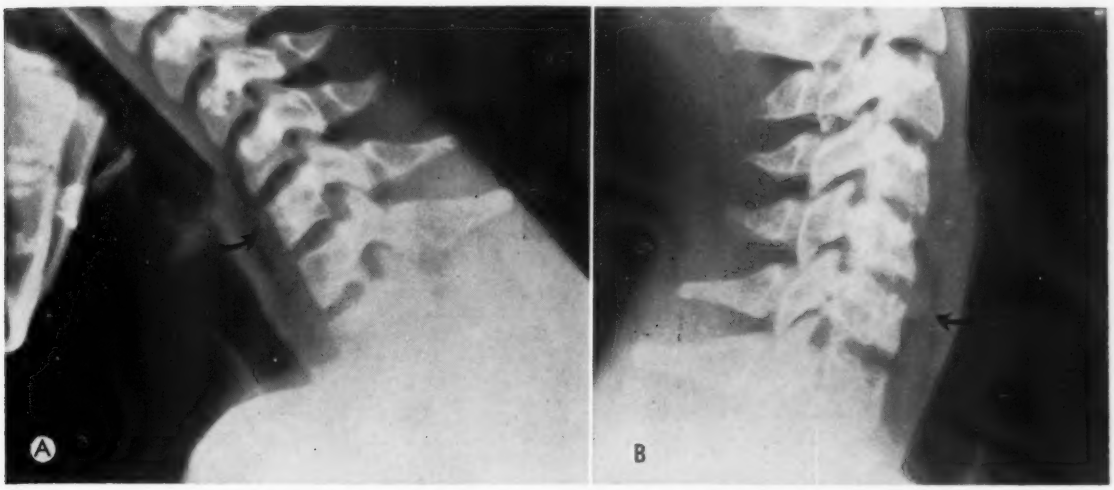

Fig. 3. Case 1. (*A* and *B*) Motion studies again show the bony projection with maintenance of its relationship to the vertebral body in flexion and extension.

in anatomic specimens showed that this bony projection is an elongation of the anterior tubercle of the transverse process of the sixth cervical vertebra. Lateral stereoscopic views of the cervical spine corroborated this.

The transverse processes of the cervical vertebrae are composed of anterior and posterior portions. The anterior portion is the homologue of the thoracic ribs and is considered an embryologic costal element.[2] The posterior portion is the true transverse process and corresponds to those structures in the remainder of the spine. The portions of the transverse process are demonstrated in Figure 4.

A transverse foramen of some degree is usually present in the upper six cervical transverse processes, giving passage to the vertebral artery, vein, and a sympathetic nerve plexus accompanying these vessels. Occasionally, the foramen in the sixth cervical vertebra is small or absent, as in the specimen shown in Figure 4. In this case the artery passes anterior to the entire transverse process.

Roentgenographic comparison of the vertebral bodies from the third cervical to the first thoracic level as shown in Figure 5 demonstrates the increasing length and anterior direction of the anterior tubercle of the transverse processes below the third cervical vertebra. The most prominent one is at the sixth cervical vertebra. It can be seen that the anterior tubercle actually protrudes anterior to the body of the vertebra at that level in the disarticulated skeleton, although this is not as marked as in the cases presented.

The anterior costal element is almost completely nonexistent in the seventh cervical vertebra and at the first thoracic level a true rib is ordinarily present. Persistence of the costal element at the seventh cervical level is often seen as a cervical rib

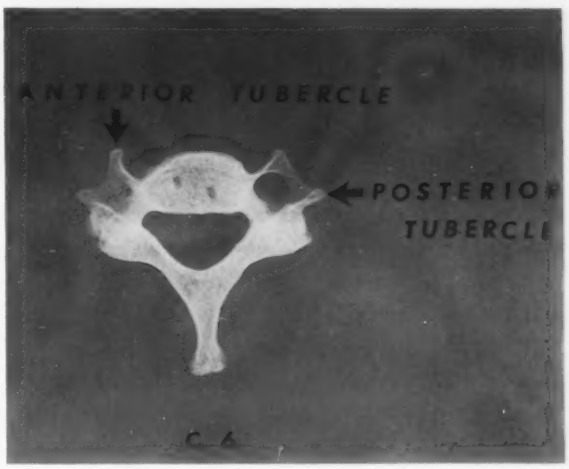

Fig. 4. Superior aspect of the sixth cervical vertebra showing the anterior and medial curvature of the anterior tubercle with projection of its most distal point anterior to the body of the vertebra.

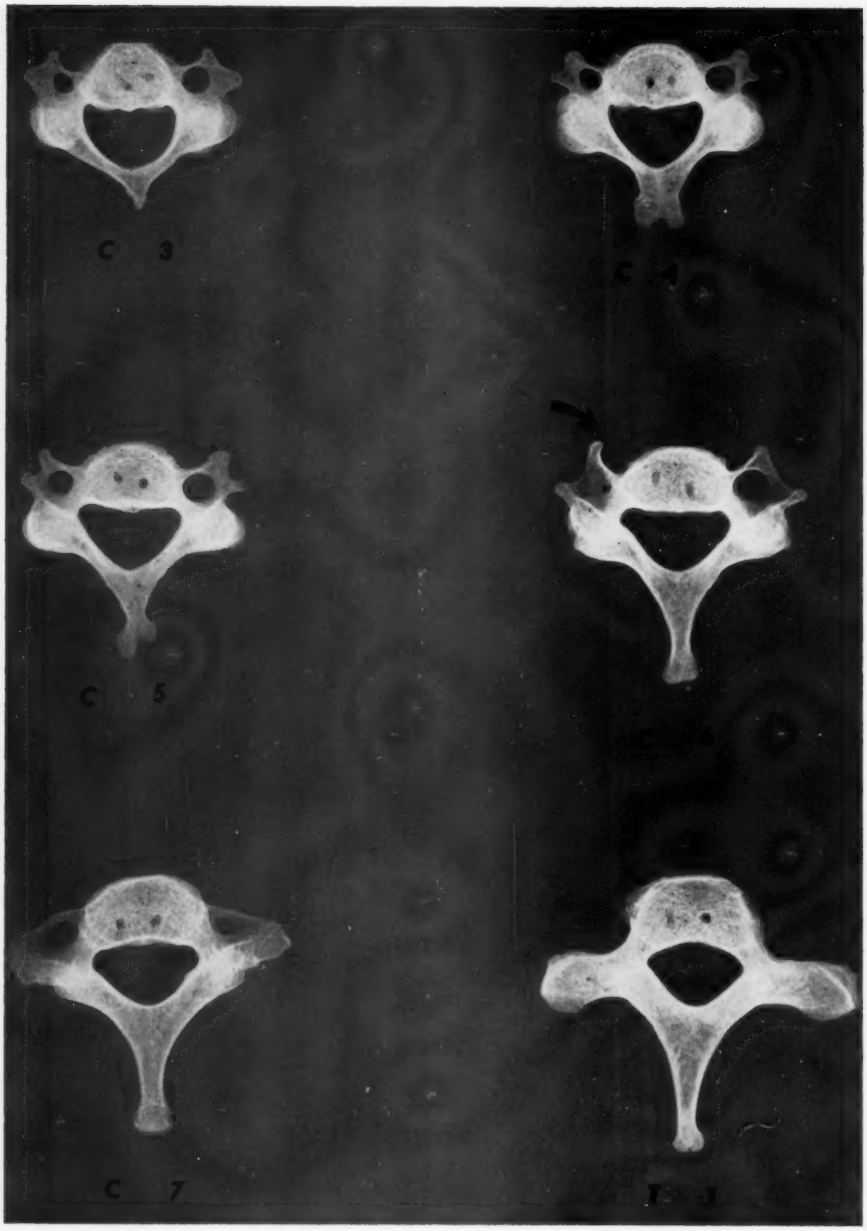

Fig. 5. Comparative appearance of the superior aspect of the vertebrae from the third cervical to the first thoracic level. The anterior tubercle of the sixth cervical vertebra is the most prominent.

with a true articulation between it and the vertebral body.

The unusual elongation of the anterior tubercle of the transverse process of the sixth cervical vertebra is independent of either elongation of the transverse process of the seventh cervical vertebra or the presence of a cervical rib.

Cineroentgenographic studies were performed using an articulated spine. Because of the comparatively faint radiodensity of the transverse process as compared with the vertebral body, lead markers were placed on the furthest projections of the anterior and posterior tubercles. Figure 6 was made from selected frames illustrating the position of the anterior and posterior tubercles of the transverse process of the

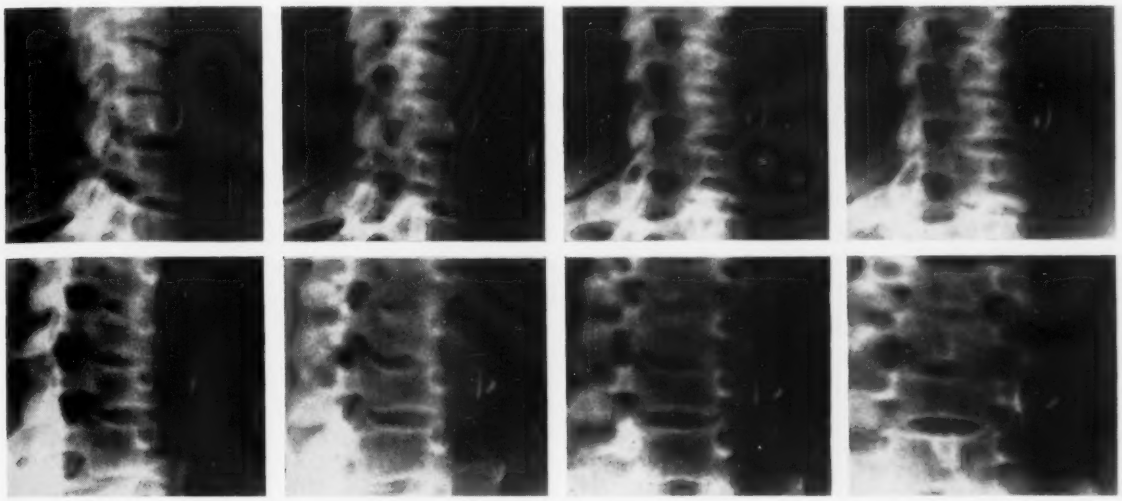

Fig. 6. Frames 1 (top, left) to 8 (bottom, right) demonstrate positions during rotation from the lateral to the posteroanterior projection. The long, lead, vertical strip is on the anterior tubercle and the short oblique marker on the posterior tubercle. Frame 1 shows the anterior tubercle projecting anterior to the vertebral body. Frame 5 shows superimposition of the tubercles in the usual oblique position. Frame 8 shows the positions in the posteroanterior projection with the anterior tubercle seen medially.

sixth cervical vertebra while the spine was rotated from the lateral to the posteroanterior projection. In the lateral view (Fig. 6, frame 1), the anterior tubercle, marked by the long vertical lead strip, is seen projecting anterior to the body of the vertebra. The posterior tubercle, marked with a short oblique strip, is superimposed on the body. It can be seen that with minimal variations in obliquity and tubercle length, either the anterior or posterior tubercle can be projected more laterally in the usual oblique view of the cervical spine (Fig. 6, frames 4-5-6). In the posteroanterior view (Fig. 6, frame 8), the distal point of the anterior tubercle can be seen medially, near the body of the vertebra.

SUMMARY

The identity of a bony projection seen anterior to the body of the sixth cervical vertebra in the lateral view of the cervical spine has been shown by routine and cineroentgenographic studies of the normal cervical spine and the skeleton.

This normal variant is an unusual elongation of the anterior tubercle of the transverse process of the sixth cervical vertebra. The knowledge of this configuration may be of considerable importance in cases of trauma or of medico-legal interest.

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The author wishes to express his appreciation to Dr. Michael B. Dooley of Phoenixville, Pennsylvania, for permission to use the roentgenograms of Case 1 and to our Medical Illustrators, Mr. Robert David and Mr. Robert Morris.

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UNILATERAL THORACIC SPONDYLOSIS*

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FOR a number of years, the senior author has been intrigued by the frequent tendency for hypertrophic thoracic spurring to be predominantly, or occasionally entirely, unilateral. The osseous spurs are usually right sided and are more common in hypersthenic, middle-aged or elderly patients (Fig. 1). Pathologically, the process consists of ossification of the lateral portion of the anterior longitudinal ligament. In its most florid form, the undulating irregularity along the lateral margin of the spine is reminiscent of the corrugations of a wash-board. The appearance of the spine has also been likened to a string of beads.[4]

FIG. 1. Roentgenogram of a patient with a normal left descending aorta in the paravertebral position demonstrating right unilateral thoracic spondylosis.

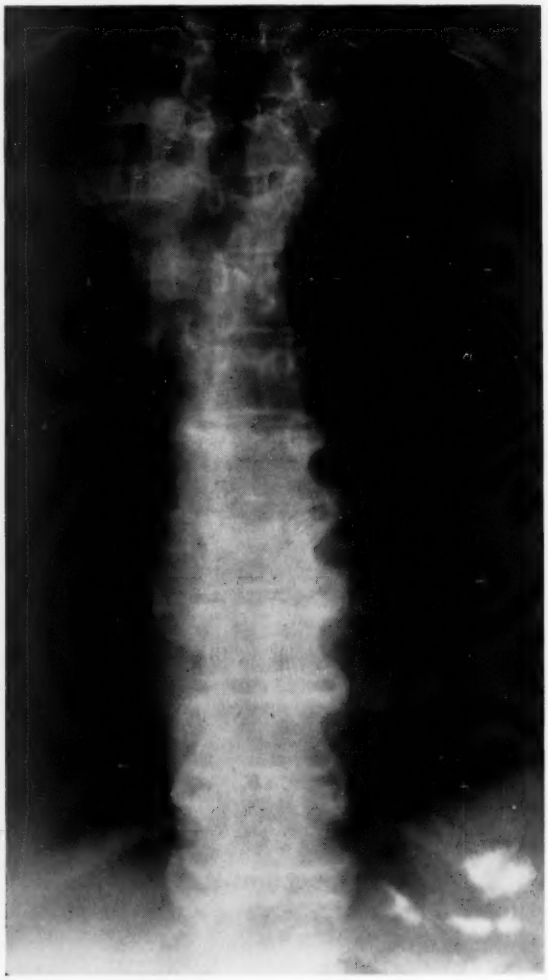

FIG. 2. In the case reported in the present paper a right aortic arch with the descending aorta in a right paravertebral position and left unilateral spondylosis down to T10 are demonstrated.

A number of theories have been advanced to explain the predominant right sided localization of the spurs. Beneke and Simmonds[1] suggested that right handedness

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Since submitting this paper the authors have come across an interesting report on this subject by Culver, G. J., and Pirson, H. S., Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1956, *76*, 1157–1160.

Fig. 3. Same patient as in Figure 2. Barium sulfate outlines the course of the esophagus.

tends to promote intervertebral disk trauma on the dominant side with secondary osteophyte formation. Meyer and Forster[2] similarly believed that right handed people tend to have a physiologic dextroscoliosis which subjects the right half of the anterior longitudinal ligament to constant minor strain. In this regard, it is interesting to note that Schmorl and Junghanns[3] reported a few left handed patients with predominant left sided spurs.

The present authors are impressed with the position of the aorta in relationship to these changes. When the abdominal aorta is in a left paravertebral location, lateral roentgenograms of the lumbar spine often demonstrate some erosion of the left anterolateral aspect of several lumbar vertebrae. These changes cannot be recognized when the aorta lies directly anterior to the spine in the midline prevertebral position. Similarly, patients exhibiting right unilateral thoracic spondylosis have a descending aorta in the left paravertebral position while patients with extensive, fairly symmetric, bilateral thoracic spondylosis have a descending aorta in the prevertebral location more or less in the midline. Perhaps then, unilateral spurring occurs on the side opposite to a paravertebral descending aorta because the normal aortic pulsations tend to produce physiologic resorption of bone. In line with this hypothesis, the authors conjectured that a patient with a right sided paravertebral aorta and thoracic spondylosis should have left unilateral spurring. Eventually such a patient appeared and turned out to have the left sided spurring predicted.

REPORT OF A CASE

B.S., a fifty-seven year old white, married housewife was seen in August, 1958, for biopsy of a mass in the left breast. The past medical history and systemic review were unremarkable. The patient was ambidextrous but favored the right hand. Physical examination was negative except for a flat, doughy mass in the left breast. The blood pressure was 120/80, and the pulse rate 76.

Fluoroscopic and roentgenographic studies showed a right aortic arch with a right descending aorta in a paravertebral position down to the level of T10 where the aorta began to cross the midline. Osteophytes were limited to the left side from T5 to T10 while at T10 to T11 bilateral spurs were found (Fig. 2 and 3). In essence, as long as the right descending thoracic aorta was paravertebral in position, the osteophytes were confined to the left side. Bilateral spurring occurred at the site where the aorta departed from its paravertebral course to cross the midline.

SUMMARY

1. A brief review of unilateral thoracic spondylosis is presented.

2. A case report of a right sided aortic

arch with left unilateral thoracic spondylosis is reported. The possible role of the position of the descending aorta in relationship to the spurring is discussed.

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THE WEIGHT FORCE IN CHARCOT DISEASE OF JOINTS: THE APPARENT PARADOX OF TIBIOFIBULAR JOINT FUSION*

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THE thing-in-being of neuropathic or Charcot's disease of any joint is its weight-ordered destruction which is seldom stayed by the hypertrophic biologic reaction. A similar weight-ordered destruction of the knee without overproduction of bone is seen in the atrophic form of the neuropathic joint or in rheumatoid arthritis. Certainly, no one has ever seen a spontaneous healing fusion of the Charcot joint. An apparent paradoxical fusion may take place in the upper or lower tibiofibular joint, but not in both.

The normal fibula is not weight-loaded for the weight force passes through the tibia. The bone is the analogue of the radius but it cannot turn about the tibia except in the unique circumstance of pseudarthrosis of the tibia. Either tibiofibular joint is seldom afflicted by the illness of its major companion joint. The tibiofibular joints are never in themselves neuropathic.

The weight force, W, (Fig. 1) is a vector directed vertically downward to the earth. It is the resultant of the infinite parallel "gravity forces acting on the various parts of the body" (Timoshenko and Young[8]). It is resisted by the equal, opposite and colinear counterweight force which is the upthrust of the earth.

When W strikes the weight-bearing joint surface obliquely, it is resolved into three mutually perpendicular and concurrent components, the resultant of which is W itself (Fig. 1). The two components in the oblique horizontal plane, W_F and W_{F1}, are shear or frictional forces whose maximal angle of application cannot exceed 45 degrees. The oblique vertical component of W is W_V, which strikes the oblique joint surface perpendicularly to it.

In Charcot's disease of the knee (in valgus) or in Charcot's disease of the ankle, W is displaced laterally and anteriorly. W_F is the shear force which cuts an oblique sur-

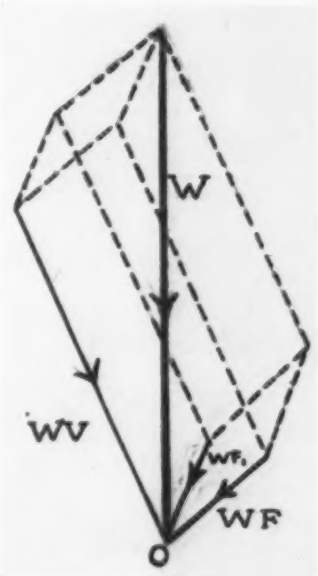

FIG. 1. A diagram to show the 3 mutually perpendicular and concurrent components of W and the parallelepiped of force when the weight force, applied anteriorly, strikes an oblique surface. W is the resultant of the infinite parallel elements of the gravity force acting on the infinite particles of the body. It is physiologically healthy when it hits the joint surface at right angles. It becomes a destructive force when it hits the joint surface obliquely and allows separate action of its three principal components. The shear or sliding frictional force makes the joint surface more oblique, *but the angle of obliquity cannot exceed 45 degrees.* The coplanar force at 90 degrees to it wears away the joint surface anteriorly to which part it is directed. The oblique vertical component, W_V, makes the fibula receive more weight force. The bone thickens and apparently elongates.

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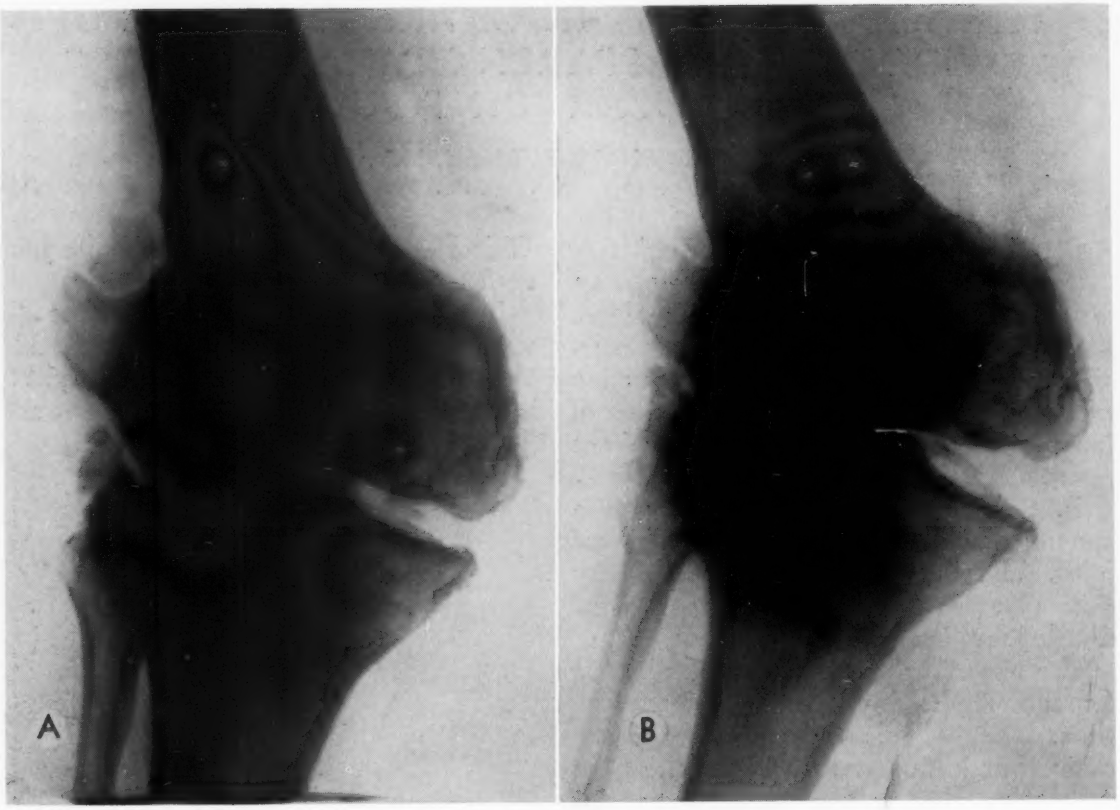

Fig. 2. (*A* and *B*) Charcot's disease of the right knee joint in a man, fifty-six years old, shown in roentgenograms taken on June 29, 1955. Destruction of the lateral tibial condyle gave valgus deformity of the knee and consequential fusion of the upper tibiofibular joint. *A* is the roentgenogram taken neutrally and *B* is the stress roentgenogram in abduction. The latter discloses the synostosis better.

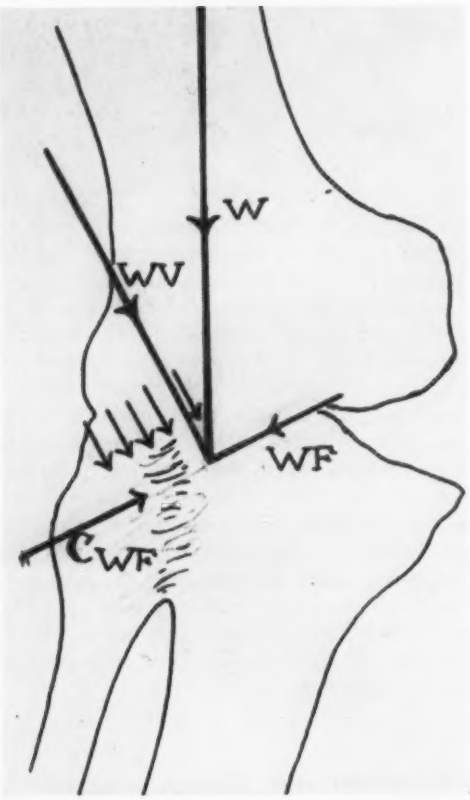

Fig. 3. This diagram shows the forces producing destruction of the lateral tibial condyle of the knee and synostosis of the upper tibiofibular joint. W_{FI} is not shown. The distributed parallel elements of W_V are depicted as they contribute to the synostosis.

Fig. 4. (*A* and *B*) Postoperative roentgenograms taken on January 14, 1959, three years after the implantation of the patella to build up the lateral tibial condyle. The patella is dense, probably by some measure of aseptic necrosis. The medial collateral ligament had been reinforced and tightened with fascia lata on May 31, 1956, six months after the operation of 1955. The disease has been stayed by the restoration of horizontality of the joint surface. The weight force is received physiologically without the development of the three adverse components of the weight force. The left knee is osteoarthritic but not neuropathic. The man wears a brace. The use of the patella was easier and more physiologic than the use of a vitallium mold in the presence of fusion of the proximal tibiofibular joint.

face from an articular surface which was previously horizontal and had received W perpendicularly. Obliquity of the lateral tibial condyle gives valgus of the knee and secondary relaxation of the medial collateral ligament of the knee (Fig. 2, *A* and *B*). In the ankle there follows varus of the foot (Fig. 5, *A* and *B*).

The other coplanar component, W_{F1}, acts in the anteroposterior direction. It rubs away the anterior half of the bone and gives recurvatum of the knee and of the ankle as the frictional consequence of the anterior displacement of the line of gravity. For this reason, one does not see flexion deformity of the knee or equinus deformity of the foot in neuropathic disease of these joints. In the ankle, the arc of motion is shifted to the field of dorsiflexion and in the knee to the field of overextension.

W_V, the oblique vertical component of W, meets W_F at a right angle. This force passes through the tibiofibular joint and is resisted by the equal and opposite force of joint constraint which holds the tibia and fibula together. These equal and opposite forces give compression of the tibiofibular joint and, in time, fusion of the joint and the adjacent area, for W_v need not be pin-

Fig. 5. Advanced Charcot's disease of the right ankle in a Chinese man, sixty years old. (*A*) Roentgenograms of the ankle alone were taken on June 4, 1958 and (*B*) roentgenograms of the whole leg, including both tibiofibular joints, a few days earlier on May 31, 1958. In the lateral view the fibula is somewhat enlarged, posteriorly bowed in the mid-part, and medially displaced to the tibia.

pointed to the joint. This fusion is an effect of the diverted force of weight. The fibular shaft thickens when it receives weight. W_F and C_{WF} as well as W_{F1} and C_{WF1} compress the tibiofibular joint to bring about this paradox of synostosis (Fig. 3 and 6).

The resultant of the coplanar forces, W_F and W_{F1}, either passes through the center of motion of the joint or it does not. If it does, there is no moment and no rotation. If it does not—and this is more likely—the moment tends to rotate the leg in Charcot's disease of the knee in valgus or the foot in Charcot's disease of the ankle. The direction of rotation is "downhill" or medially. This is resisted by the synostosis.

The single resultant force is equivalent to a force couple of equal moment. The turn takes place about an axis (moment vector) perpendicular to the inclined articular surface, an axis collinear with W_v or parallel to it.

Fusion of the upper tibiofibular joint is illustrated by Figures 2 and 4, and of the lower tibiofibular joint in Figures 5, 7 and 8. In Charcot's disease of the ankle, there is an increasing lateral displacement of W with increasing varus. The fibula hypertrophies and elongates distally to the outer

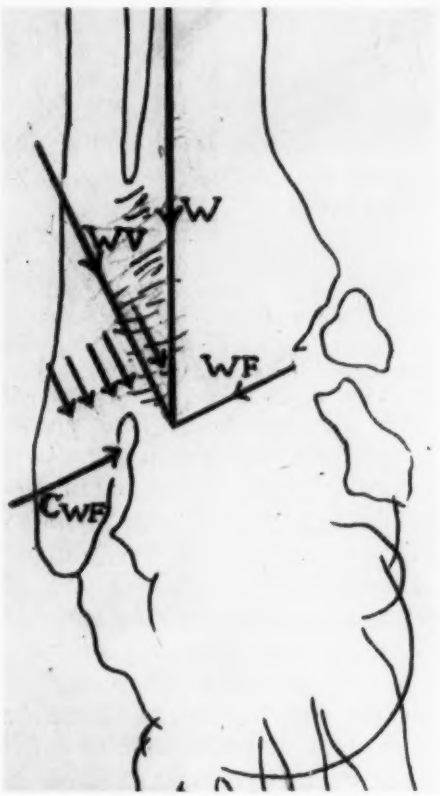

Fig. 6. This diagram, as in Figure 3, reveals the forces destroying the ankle joint and giving synostosis of the lower tibiofibular joint.

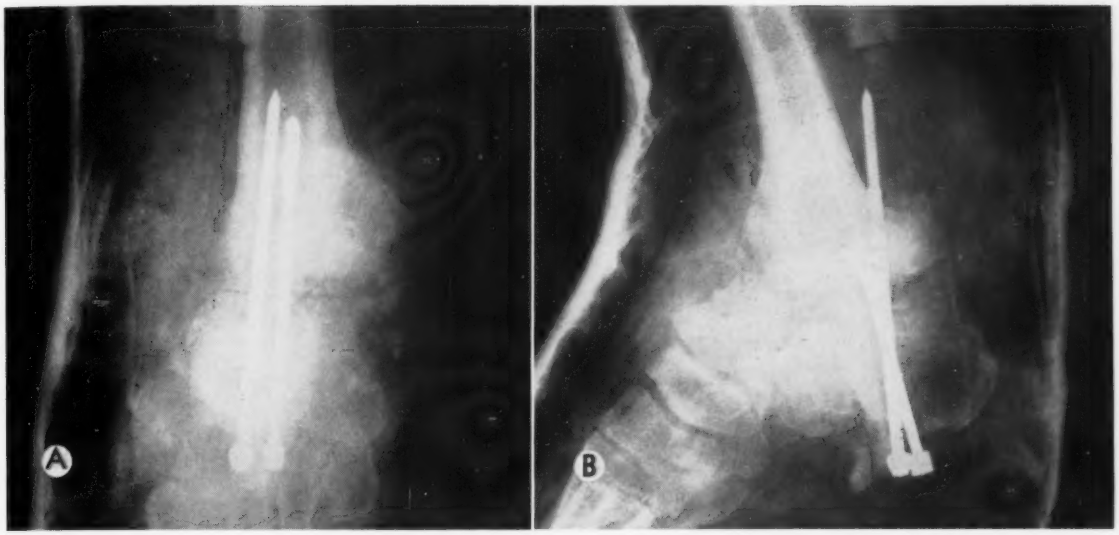

Fig. 7. (*A* and *B*) Anteroposterior and lateral postoperative roentgenograms taken on June 14, 1958. The greater part of the astragalus had been resected, excepting its head and neck. The new joint line of tibia to heel bone was made horizontal to receive W perpendicularly. Transfibular fusion had been planned but, after astragalectomy, there was insufficient purchase of the graft on the outer side of the heel. An intramedullary form of fixation was used, reinforced by two Knowles pins. This gave solid fixation. The removal of the fibular graft was made difficult by the extent of the fusion of the fibula to the tibia over a distance of two or more inches.

side of the heel bone. Fusion between the tibia and fibula exceeds the area of the quondam lower joint.

The absolute or bony fixation of the fibula below (this means its medial movement to the tibia) and relative fixation of the fibula above at its joint with the tibia allows posterior bend of the middle part of the fibula. (Fusion of the fibula to the tibia may take place in a Charcot ankle when the lower end of the fibula is broken and the talus laterally displaced, without significant obliquity of the joint line. The medially placed stump of the proximal fragment is joined to the tibia by overabundant callus. This was shown in Figure 3c of the paper by Kelly and Coventry.[6])

The knee and ankle joints are sometimes simultaneously afflicted in Charcot's disease. Yet the simultaneous fusion of the two tibiofibular joints is mechanically denied. This demands the association of valgus of the knee and varus of the foot.

If tibiofibular joint fusion does not always take place, either the angle of shear is not maximal or there is scanty biologic preparation for fusion. There may be lack of the not uncommon parallel periostitis of bone in Charcot's disease, "entorse" (twist) of the tibiofibular joint or fracture in or near the joints as events give background for fusion.

COMMENT

The observation of fusion of the tibiofibular joint does not reveal an easier way to get fusion of a Charcot joint, for the tibiofibular joint is not itself neuropathic. It spells out the importance of the weight force directed to salutary compression and its possible application to fusion of the Charcot joint. More immediate is the surgical fact that the fusion of the upper tibiofibular joint may block reconstruction of the lateral tibial condyle. The fusion of the lower tibiofibular joint is to be reckoned with in transfibular fusion of the ankle or any fusion in which the lower end of the fibula is used as a graft.

ADDENDUM

A synostosis between the lower ends of the tibia and the fibula may take place after fracture of the ankle or of both bones in

Fig. 8. It was expected that fusion would take place. It did not, for the man began walking on the plaster cast against instruction. The graft broke. (*A* and *B*) Roentgenograms taken on July 1, 1958 showed disintegration, fracture of the graft and separation of the heel bone from the tibia. The sequestrated graft was removed through the infected stab wound in the heel. A new plaster cast was applied and he was transferred to the Psychiatric Division of Bellevue Hospital for an increasing mental disturbance with confusion and suicidal tendency, said by them to be arteriosclerotic in origin. He recovered quickly from this to be discharged into his own custody on September 4, 1958. The heel wound healed quickly. The last roentgenogram taken on August 18, 1958 revealed no fusion.

their lower fourths before there is known Charcot arthropathy on the same or other side or in known same-sided arthropathy, not necessarily in the ankle. The synostosis is caused by an excessive periosteal reaction giving abundant callus for there is the proprioceptive lack of tabes dorsalis. The synostosis is not the effect of the paradox described above.

I have seen this in the following two situations—when there was no Charcot disease of the ankle and when there was such disease: (1) The leg is broken in its distal fourth. The ankle joint is normal and the same-sided Charcot knee joint is deformed in varus. This situation was found in a Negro male in his fifties. Both leg bones had been broken at equal level in

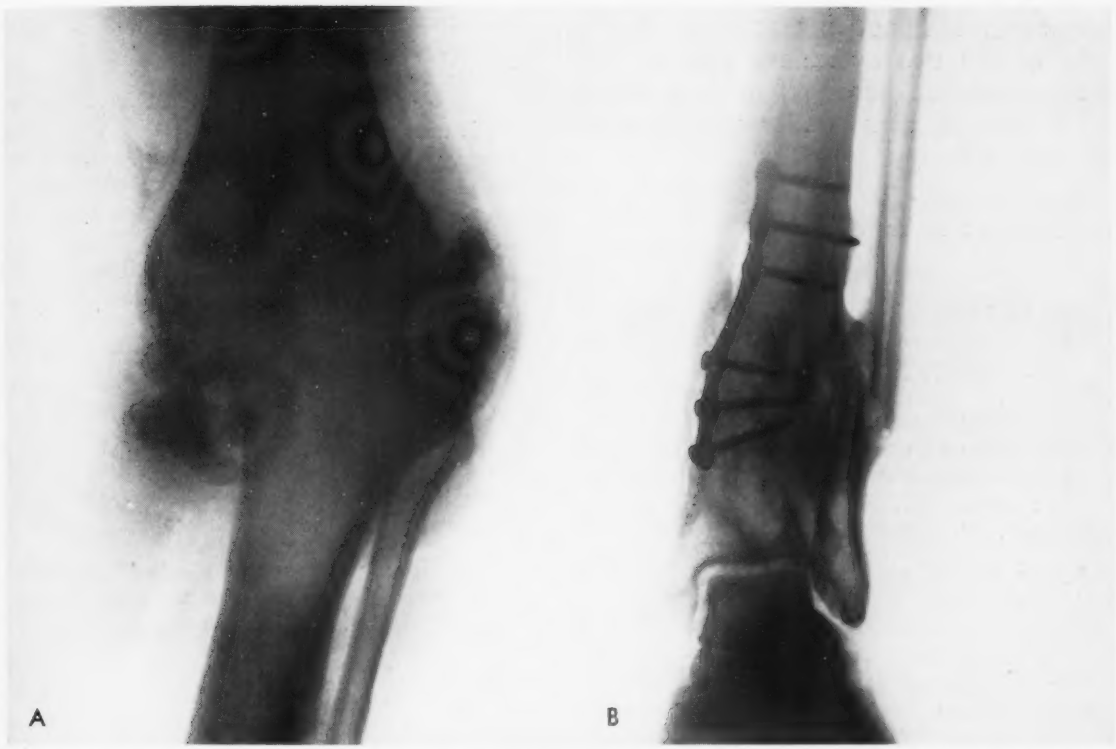

Fig. 9. (*A*) Anteroposterior roentgenogram taken on January 5, 1949. Charcot's disease of the left knee joint in a Negro male in his middle fifties. There is great destruction of the medial tibial condyle and consequential bowing of the joint into varus. The angle of shear is not maximal. (*B*) A great trauma broke both bones of the left leg in their lower fourth. The tibia was plated and six screws held the plate to the tibia. The fibula broke at equal level. Its distal fragment was displaced medially and fused to the tibia for its entire length. The laterally displaced proximal fragment was fused to the tibia and to the distal fragment over a more limited area. The ankle joint was normal. This is the anteroposterior roentgenogram of the left leg and ankle taken on the same day.

their lower fourth in 1947. A fusion of the tibia to the fibula took place. Bone plating itself need not favor an exuberant callus. The opposite or right ankle was the site of an advanced Charcot disease. Did the bowing of the knee induce synostosis? Probably not, for the limb was neutrally held in plaster and weight-bearing was not allowed until healing took place (Fig. 9, *A* and *B*). (2) The ankle joint is broken. The fracture seemed to antedate the Charcot arthropathy in the same joint. There is an opposite-sided Charcot knee joint. This happened to a Negro woman who broke her right ankle in 1947 and underwent two operations then to set it. At some unknown later time, the right ankle became deformed by Charcot disease, and so too did the left knee. She is now fifty-nine years old. The right ankle is in *neutral* position. There is a synostosis of the tibia to the fibula in their most distal parts for almost two inches. The fibula is thickened by parallel periostitis for almost all its length excepting its most proximal part. The right knee and the upper tibiofibular joint are normal. The lower half of the fibula is posteriorly bowed a little. There has been no (recent) progression of arthropathy. It may well be that in 1947 she had tabes dorsalis and that the synostosis existed long before the coming of the Charcot arthropathy. Did the fracture of the ankle localize the neuropathic disease to the ankle? Did not the synostosis between tibia and fibula hinder the advance of the Charcot disease by holding the mortise even though the body of the talus dis-

integrated? Did not the scarring in the wake of the two operations prevent the rotary deviation of the foot into varus which now in truth under weight is in valgus?

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LITTLE LEAGUER'S ELBOW*

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APPARENTLY one of the more hazardous of summertime occupations is that of throwing a baseball. The old post-traumatic, hypertrophic, arthritic elbow of the veteran pitcher is well described and familiar.[7] Dotter[3] has written on "little leaguer's shoulder," a fracture of the proximal epiphyseal cartilage of the humerus. Several cases are reported in which adults sustained a fracture of the humerus while throwing a ball with maximum effort.[1,4,9]

During the past baseball season we have seen in pubescent pitchers an injury which we choose to call "little leaguer's elbow." The similarity of these cases is striking.

REPORT OF CASES

CASE I. C.S., a well-developed eleven year old fast-baller, pitched the opening game of the Little League season. Despite an inadequate warm-up he pitched a no-hitter and allowed only one walk! However, by the middle of the game he was experiencing pain over the medial epicondyle of the pitching arm with every throw and could not really "bear down." For several days afterward there was local tenderness and swelling over the right medial epicondyle and pain on extreme flexion or pronation of the forearm. He vacationed for one month and did not pitch or otherwise violently exercise the elbow. The patient was brought to the hospital by his father when attempted resumption of his pitching career reproduced the initial symptoms.

Roentgenographic study of the elbow (Fig. 1, *A* and *B*) revealed separation and fragmentation of the epiphysis of the right medial epicondyle and loss of fascial markings about the elbow suggesting edema or hematoma.

The patient was forbidden to pitch or otherwise strenuously exercise the elbow. One month later all symptoms had disappeared, but a clandestine experiment on the part of the patient had proved that pitching still elicited pain. Repeat roentgenograms showed the epicondylar epiphysis to be in good position, albeit slightly separated, with no callus formation about the epicondyle.

CASE II. L.C. was a muscular twelve year old right-hander whose delivery was stylized by a marked wrist-flexing terminal snap which was supposed to add speed to the ball. During a Little League game early in the season he threw a maximum effort fast-ball and immediately sustained severe pain in the right elbow which necessitated his leaving the game.

Tenderness and swelling over the medial epicondyle with limitation of elbow flexion developed. These symptoms persisted, and the patient came to the outpatient department.

Roentgenographic examination of both elbows (Fig. 2, *A* and *B*) revealed separation of the epiphysis of the right medial epicondyle and adjacent soft tissue swelling.

The patient was instructed to restrain from further pitching and to rest the elbow as much as possible. This treatment was efficacious, and three weeks later all symptoms had disappeared. Re-examination of the elbow showed no change in position of the epiphysis and no evidence of callus formation.

Upon the advent of the junior high football season the patient was advised to become a place-kicker.

CASE III. T.R. was a right-handed thirteen year old Pony League pitcher who was being groomed by his father for a big league career. In midseason he entered a game, without warming up, as a relief pitcher. On the first pitch, a fast-ball, the patient experienced severe pain in the right elbow and rapidly developed swelling and tenderness over the medial epicondyle. There was pain on elbow flexion producing a 15 degree limitation of motion. Five days later signs and symptons had not regressed, and the patient's father brought him to the hospital.

Comparison roentgenograms of the elbows (Fig. 3, *A* and *B*) demonstrated avulsion and fragmentation of the right medial epicondylar epiphysis and surrounding soft tissue swelling.

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Fig. 1. Case 1. (*A*) Right elbow demonstrating avulsion and fragmentation of the ossification center of the medial epicondyle and adjacent soft tissue swelling. (*B*) Normal left elbow.

The patient was operated on, and the epiphysis was fixed in place with two threaded Kirschner wires. The elbow was immobilized in flexion.

Examination one month later revealed no tenderness or swelling and the incision was well healed. Roentgenograms showed the epiphysis to be in excellent position. There was minimal periosteal callus proliferation about the medial epicondyle. The elbow was placed in a sling for two additional weeks with subsequent gradual resumption of active motion.

DISCUSSION

MECHANISM OF INJURY

Avulsion of the ossification center of the medial epicondyle may be sustained in various ways. The mechanism of avulsion is tension on the common tendon of the wrist flexors, finger flexors, and the pronator teres. Displacement is always in a downward direction.[5] This injury may be induced by hyperextension of the hand and elbow. However, in our cases we believe separation to have been caused by tension on the common tendon resulting from extremely vigorous contraction of the flexor-pronator group.

Analysis of the mechanism of pitching reveals the following motions (Fig. 4): At the end of the windup, the arm is extended and externally rotated; the elbow is almost completely extended and the hand is extended; the arm is then forcefully and rapidly flexed, adducted and internally rotated; the elbow is flexed and re-extended while the forearm is pronated and the wrist is sharply flexed in a terminal snap.

The inexperienced or poorly coordinated player, in an effort to achieve maximum speed or power, may hurry or snap short the motion and thus activate opposing muscle groups simultaneously. This reinforces and increases muscle tensions. Lack of proper warm-up or conditioning prior to maximum-effort pitching may contribute to incoordination.

AGE FACTOR

In Smith's collection of 137 cases of

Fig. 2. Case II. (*A*) Right elbow showing separation of medial epicondylar ossification center and surrounding soft tissue swelling. (*B*) Normal left elbow.

medial epicondyle injuries, 99 were in patients 9 to 14 years old. Average age for the entire group was 11.3 years.[6] Average age of 11.7 years was found in another similar, but smaller, series.[2]

These findings suggest that Little Leaguers (ages 9 to 12) and Pony Leaguers (ages 13 to 14) are placed on a pitcher's mound, inspired to maximum efforts by the adulation of their contemporaries and the exhortations of their often vicariously ambitious parents, at the worst possible time in their lives—at least insofar as the medial epicondyle is concerned.

ROENTGENOGRAPHIC CONSIDERATIONS

Epiphyseal trauma without roentgenographically apparent separation may occur, the diagnosis being clinical.[5,6] Demonstrable epiphyseal separation may be associated with fragmentation of the ossification center or, in cases of violent motion, dislocation of the elbow joint. Soft tissue injury will be evident. Comparative roentgenograms of the contralateral elbow are extremely helpful in diagnosing minimal separation and should be obtained in every case. Since the epiphysis tends to displace inferiorly, it sometimes enters the ulnotrochlear joint. Epicondylar separation treated by closed reduction commonly heals by fibrous union. The common flexors gain a new humeral attachment through scar tissue.[6,8] Consequently, roentgenologically demonstrable callus is not encountered. The callus which developed in Case III was probably the result of surgical intervention.

Many minimal cases probably are never clinically or roentgenologically examined, being considered and treated at home as "pulled muscles" or "sore pitching arms."

THERAPY

Most orthopedic texts advise treating minimal separations by closed manipulation and immobilization for several weeks with the forearm flexed and partially pro-

Fig. 3. Case III. (*A*) Comparison of both elbows demonstrates separation and fragmentation of the ossification center of the right medial epicondyle. There are changes in the adjacent soft tissues suggesting edema or hematoma. (*B*) The left elbow is normal.

nated. This is followed by partial rest in a sling with gradual resumption of active motion. Passive exercise and stretching are not advised.

There is a good reason for argument that these injuries are often overtreated.[6] Delay or failure in the return of full elbow extension is sometimes encountered and may be related to prolonged splinting in elbow flexion. Smith[6] advises that the use of splints and slings be limited to a short period of time and, in effect, that the pa-

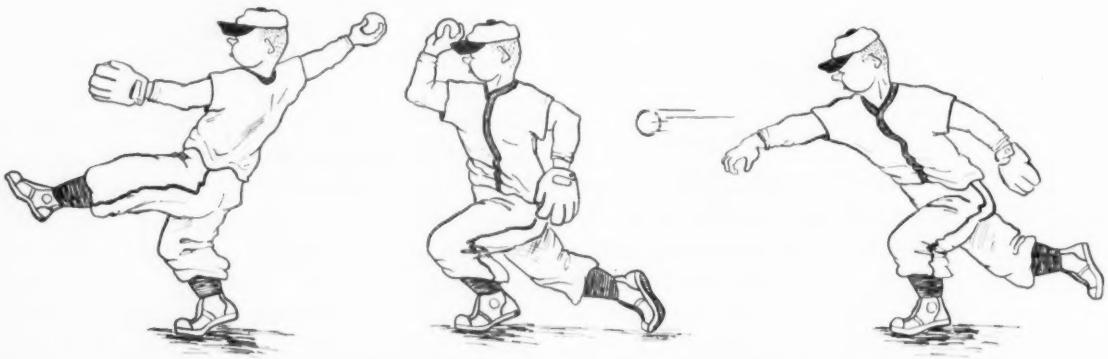

Fig. 4. The pitching motion shown (left to right) at the end of the windup, in mid-delivery, and immediately following release of the ball.

tients be treated symptomatically. Our Case I and Case II have done well thus far with voluntary limitation of elbow activity.

Wide separations and displacements of the medial epicondyle are treated with open reduction and, usually, internal fixation.

SUMMARY

Three cases of "little leaguer's elbow," an avulsion of the ossification center of the medial epicondylar epiphysis in pubertal pitchers, are described. In all cases the separation appeared to result from the violent muscular activity of throwing a baseball with maximum effort. The mechanism, age factor, diagnosis, and treatment of this injury are discussed.

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FRACTURES OF THE TRIQUETRUM

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FRACTURES of the triquetrum form the second most common group of carpal injuries; they are, nevertheless, uncommon as compared with navicular fractures. Of 175 fractures of the carpal bones, Kellam and McGoey[4] found 11 triquetral fractures (6 per cent). Bonnin and Greening[2] in their series of 580 wrist injuries found 20 fractures of the triquetrum (3.5 per cent). Undoubtedly the true incidence is higher than these figures indicate, since small chip fractures of the dorsum are frequently missed or mistakenly called fractures of the posterior pole of the lunate. Actually, this latter type fracture is exceedingly rare, accounting for less than 1 per cent of all carpal injuries and can be readily differentiated from triquetral fractures on roentgenograms.

Frequently, triquetral injuries are associated with more important fractures of the navicular bone or radius or with semilunar dislocations. Thirty per cent of 46 triquetral fractures in the series of Bartone and Grieco[1] had associated injuries.

The mode of trauma varies. Direct injury to the triquetrum is rare, since it is protected by the overhanging edge of the ulna. Occasionally a direct blow fractures the dorsum. Usually, the injury results from a backward fall on the outstretched hands. Either violent dorsiflexion with ulnar deviation or forcible palmar flexion occurs, causing compression of the triquetrum against the lunate, pisiform, or lower end of the radius or ulna. Infrequently, sudden palmar flexion and radial deviation of the hands produce an avulsion fracture of the tuberosity with separation and sprain of the ulnar collateral ligament. Usually, however, this type of trauma causes a fracture of the ulnar styloid. Rarely, unless by direct trauma, do fractures of the ulnar styloid and triquetrum coexist, since the ulnar collateral ligament, attached to both bones, separates at only one point.

Specific physical findings and signs may be masked by associated injuries of the wrist. As is to be expected, fractures of the triquetrum are followed by pain, swelling, tenderness, and restricted movement of the wrist. The classic fracture, that of the posterior tuberosity or dorsum, shows swelling localized to the ulnar side of the wrist, local tenderness maximum over the tubercle, and moderate pain on all movements of the wrist, particularly those of palmar flexion and radial deviation. The fracture fragments are rarely palpable.

Roentgenographic examination of the wrist is diagnostic and reveals two types of fractures. Small linear fissures or comminuted fractures of the body of the triquetrum are rare and usually associated with the more common flake or chip fractures of the dorsum. Bartone and Grieco[1] in their series of 46 cases found only one instance of an isolated fracture of the body and 8 examples of fractures of the body associated with avulsion or chip fractures of the dorsum. Frequently overlooked or missed, fractures of the body of the triquetrum are undisplaced, heal without treatment or complications, and never cause functional impairment. Their recognition depends upon a high index of suspicion and careful roentgenographic examination. The posteroanterior view with the wrist in radial deviation is the optimum position to reveal the fracture line, separating the normally superimposed triquetrum from the pisiform. However, oblique views are frequently needed to confirm the presence of a suspicious fracture.

Fractures of the dorsal surface of the tri-

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Fig. 1. (*A*) No fracture seen. (*B*) Lateral projection shows a fracture of the dorsum of the triquetrum.

Fig. 2. Fractures of the dorsum of the triquetrum and of the radius. (*A*) The oblique view, separating the dorsal surfaces of the lunate and triquetrum, shows the site of the triquetral fracture. (*B*) The lateral projection shows the separation of the fragment, but not the site of the fracture.

Fig. 3. (*A*) February 3, 1958: No fracture seen. (*B*) February 3, 1958: A small avulsion fracture of the dorsum is demonstrated. (*C*) Oblique and (*D*) lateral views fourteen months later show a healed fracture with deformity. Functional recovery was complete.

quetrum in tha nature of chip, flake, traction, or avulsion fragments comprise over 90 per cent of all triquetral injuries.[3] Best observed in lateral and oblique roentgenograms of the wrist and never seen in the posteroanterior view, they appear as single or multiple fragments varying in size from a millimeter to over a centimeter (Fig. 1, 2 and 3). These fragments may be undisplaced or lying free, parallel to the dorsal surface of the bone, simulating the appearance of a sesamoid. The lateral roentgenogram of the wrist shows the fracture fragments but not always the site of the fracture. In this view it is not always easy to tell whether the fragments come from the dorsum of the triquetrum or the posterior pole of the lunate; the oblique view, separating the dorsal surfaces of these bones, shows the site of fracture in every case.

Following treatment consisting of immobilization of the wrist in a plaster cast or splint depending upon the severity of the fracture and associated injuries, the fragments may reunite with or without deformity or may remain separated, their edges rounding off. Usually complete functional recovery occurs.

Roentgenographic examination is the only means of identifying triquetral fractures. When associated with other injuries of the wrist, they are of secondary importance. By themselves they are frequently missed and called sprains. This latter diagnosis may result in improper or inadequate treatment and persistent pain with impaired function of the wrist. Prompt roentgenographic examination and recognition of the fracture followed by immobilization for a few weeks assures total functional recovery. Serial roentgenograms cannot be depended upon as a guide to therapy, because bone union either never occurs or, in most cases, is delayed well beyond the time of clinical or functional recovery. The degree of bone deformity or nonunion as seen on the roentgenogram is neither prognostic nor related to functional recovery.

SUMMARY

Fractures of the triquetrum are rare and often are associated with other injuries of the wrist. Two types occur—those of the body and those of the dorsum. The uncommon fracture of the body is usually undisplaced, heals without treatment or complication, and is best demonstrated on posteroanterior roentgenograms of the wrist in radial deviation.

Fractures of the dorsum in the form of chip, flake, traction, or avulsion fragments are best seen on oblique and lateral roentgenograms of the wrist. These fractures heal with or without deformity and may remain ununited.

The roentgen appearance of triquetral fractures cannot be used as a guide to therapy, since the degree of deformity or nonunion is unrelated to functional recovery, which is usually complete.

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ROENTGEN MANIFESTATIONS OF SYNOVIAL OSTEOCHONDROMATOSIS*

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NUMEROUS descriptions of presumed synovial osteochondromatosis in the literature have obscured the fact that this entity is rarely seen and have further compounded confusion regarding its pathogenesis and relationship to superficially similar categories.

Jaffe,[4] in his excellent book, states that the term "synovial osteochondromatosis" is properly applicable only to a condition in which cartilaginous foci develop in joint synovia through metaplastic changes of the subsynovial connective tissue. The cartilage may then separate from the synovium, enlarge in the joint space (since it can be nourished by synovial fluid), and ultimately become calcified or ossified.

Such a definition of synovial osteochondromatosis implies that unless areas of cartilaginous metaplasia can be demonstrated microscopically, the presence of calcified or ossified joint loose bodies is not diagnostically specific and may also be seen in other entities which are discussed below.

Recently, 2 cases of synovial osteochondromatosis have been observed by us and are being reported.

REPORT OF CASES

Case 1. A.R., a thirty-four year old white salesman, was admitted on Feburary 4, 1959, with a five year history of increasing swelling, stiffness and pain in the right knee. Previous treatment, which included physiotherapy, joint aspirations, and intra-articular injections of cortisone, had been ineffective. Physical examination at the time of admission revealed the right knee to be enlarged by a brawny, moderately tender swelling. There was a slight increase in warmth but no apparent effusion. Quadriceps atrophy was noted together with limitation of range of motion of the knee.

Roentgenograms (Fig. 1 and 2) demonstrated a soft tissue mass filling the suprapatellar and infrapatellar areas with extension posteriorly. Scattered areas of amorphous calcification of a rather fine nature were dispersed throughout the mass. An area of bone erosion involving the lateral submetaphyseal tibia and adjacent fibula was noted. Laminagraphic studies were performed but added little to the observations made on the plain roentgenograms.

On Feburary 5, 1959 the knee was explored through a medial parapatellar incision. A markedly thickened, congested synovium was encountered together with numerous grayish, shiny osteochondromatous structures encrusted

Fig. 1. Case 1. A water density lobulated mass involves the suprapatellar, infrapatellar and popliteal regions. Fine, stippled coalescing calcification is present.

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Fig. 2. Case 1. The oblique projection reveals a sclerotic rimmed zone of bone erosion of the lateral submetaphyseal region of the tibia and adjacent fibula.

Fig. 3. Case 1. Photograph at time of surgery. Medial femoral condyle is exposed. Patella is held by lower retractor. Nodular, shiny, cartilaginous excrescences are seen, attached to synovium and free within the knee joint. (Courtesy Dr. M. Lazansky.)

in the synovium and lying free in the joint (Fig. 3). The skeletal changes seen on the roentgenograms resulted from pressure erosion by encrusted synovium and free bodies, and not from actual invasion. Loose bodies were removed and a synovectomy was performed (Fig. 4).

Microscopic examination of the surgical specimen was reported as showing islands of actively growing cartilage partially calcified and ossified within the thickened synovial connective tissue (Fig. 5). The final diagnosis was synovial osteochondromatosis. Cultures of fluid obtained from the knee joint at surgery were negative.

The patient was discharged on the eighteenth postoperative day on crutch walking and followed in the orthopedic and rehabilitation clinics. When last seen, four months after surgery, he felt much improved and was working regularly. Roentgenograms at this time revealed a few small flecks of calcification posteriorly and in the suprapatellar region. There was persistence of the zone of bone erosion.

Case II. H.B., a forty-nine year old Negro carpenter, was admitted on October 19, 1958 with a five year history of intermittent pain and swelling of the right knee. In 1956, at another hospital, cartilage was said to have been removed from the knee. Previous physiother-

Fig. 4. Case 1. Encrusted synovium and free bodies removed at surgery. (Courtesy Dr. M. Lazansky.)

Fig. 5. Case I. Section of synovium (×35). Upper field shows thickened synovium with synovial lining cells. Immediately below are strands of collagen tissue. Below this are islands of cartilage with early calcification. Plump bilobed nuclei indicate active growth. Below the collagen on the left is the loose connective tissue stroma.

apy provided no relief. Pertinent physical findings at the time of admission included a swollen,

Fig. 6. Case II. Water density mass largely posterior and in infrapatellar region. Stippled coalescing calcification is noted.

Fig. 7. Case II. Lateral laminagram. A lobulated posterior mass is well visualized. Stippled calcification is noted. No bone erosion.

tender, warm right knee with marked limitation of range of motion but no obvious fluid.

Roentgenograms (Fig. 6 and 7) revealed a soft tissue mass in the popliteal region containing finely stippled areas of calcification. Some osteoporosis, but no bone erosion, was present. On October 23, 1959 the knee was explored through a posterior incision. A markedly thickened capsule and synovium were encountered. Grayish, knobby, lobulated bodies were present within the joint and several were removed.

Microscopic examination (Fig. 8) revealed nests of proliferating cartilage within the synovium. The final diagnosis was synovial chondromatosis. The patient was discharged on the sixteenth postoperative day. A roentgenogram at this time showed persistence of the calcification posteriorly.

CLINICAL CONSIDERATIONS

Synovial osteochondromatosis[7] is most frequently seen in adults between the ages of thirty and fifty. No age group is exempt, however, and cases are recorded at age thirteen[9] and sixty-two.[10] Three-quarters of the cases occur in males. The knee is the most common site of involvement, al-

though other joints such as the elbow, ankle, hip and shoulder are infrequently affected. Bilateral involvement is rare. Occasionally, bursae and tendon sheaths may be involved.[12]

The patient usually presents with a long-standing history of intermittent dull aches associated with limitation of motion and occasionally accompanied by locking and grating sensations. Physical findings vary with the degree and site of involvement. Joint deformity is usually apparent, fluid may be present and movable or fixed masses are often palpable. Generally, one is impressed with the paucity of interference with function despite the size of the cartilage masses.

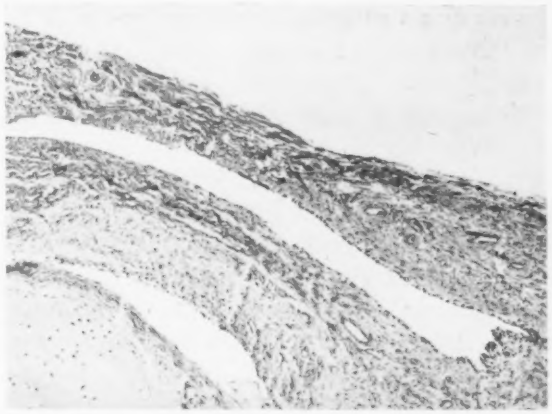

Fig. 8. Case 11. Section of synovium (×35). Synovial cleft lined by synovial cells crosses field from upper left to lower right. Lower left field shows portion of chondromatous tissue. There was no calcification or ossification in this specimen.

PATHOLOGY

Grossly, numerous flat or pedunculated, gritty, grayish nodules are attached to thickened synovial folds or communicating bursae of the involved joint (Fig. 3). Varying numbers of cartilaginous bodies, often calcified or ossified, float free within the joint cavity.

Histologically, connective tissue mesenchyme is seen adjacent to cartilage cell nests, demonstrating plumpness and double nuclei indicative of active growth (Fig. 5 and 8). Zones of calcification, ossification and even fatty marrow may be visualized.

The developing cartilage and subsequent osseous tissue are widely interpreted as stemming from metaplasia of persisting strands of primitive connective tissue mesenchyme[4] which is known to be normally present in synovium and at points of reflexion of normal joint capsules.[7] Why such embryonal connective tissue strands continue to exist in completed joint structures is not clear, nor has the mechanism of stimulation of metaplasia been explained. At various times, infection, trauma and neoplasm have been postulated.

Although the most recent hypotheses of origin favor benign neoplasia,[9] Lichtenstein's[8] conclusion is that synovial osteochondromatosis is best considered a self limited metaplastic process analogous to the local metaplasia of muscle connective tissue seen in myositis ossificans. He feels that the formed chondral or osseous bodies, while occurring in impressive numbers, have a limited growth potential. The spontaneous arrest and regression reported by Freund[3] and others would appear to support a non-neoplastic origin. On the other hand Jones[5] mentions cases with degeneration to chondrosarcoma, a concept not widely accepted.

One may conclude that disagreement still exists as to the specific pathogenetic mechanisms.

ROENTGEN FINDINGS

DIAGNOSIS

If proper roentgen techniques are utilized and due consideration is given to the clinical picture, a diagnosis of synovial osteochondromatosis will frequently be apparent.

Plain roentgenograms in frontal, lateral and oblique projections made with both soft tissue and bone exposure are often sufficient. Laminagraphy further delineates the nature of the calcifications present and often resolves the question of adjacent osseous changes. Arthrography with radiopaque media or air has been used to visualize uncalcified or unossified cartilage-encrusted synovium and free bodies. This

technique also indicates the extent of joint involvement in cases with incomplete calcification.

The roentgenographic findings are as follows:

1. Soft tissue masses of water density within the involved joint. These represent the thickened, encrusted synovium as well as free cartilaginous joint bodies and often are rounded in contour against the relatively radiolucent adjacent fat spaces.

2. Calcifications and/or ossifications representing synovial metaplastic changes to cartilage, sufficiently calcified so as to be radiopaque. These radiopacities are generally finely stippled and often coalesce to form amorphous irregularly bordered shadows which may involve the entire joint space (Fig. 1 and 2) or only a portion of it (Fig. 6 and 7). There need be no relationship between the amount of radiopaque cartilage and the extent of joint involvement by synovial metaplasia. In Freund's[3] case, for example, 395 nonopaque free bodies were removed at the time of diagnostic joint exploration. Coarser, larger calcifications, contrary to popular belief, are quite uncommon in true synovial osteochondromatosis, and are usually seen in this entity in the less frequently involved joints such as shoulders, hips and elbows.

3. Erosion of adjacent subchondral bone. This finding (Fig. 2) is related to pressure from adjacent extensively thickened synovium or free bodies and results in radiolucent areas within bone. These are rimmed with a sclerotic zone attesting to the chronicity of the change. No real destruction or subperiosteal new bone formation occurs. Bone may re-deposit in an area of erosion after surgery.[1] In our Case 1 a roentgenogram four months postoperatively showed no change in the erosive appearance.

4. Secondary arthritic changes. These result from constant mechanical trauma of the metaplastic bodies and synovium and include osteophyte formation, thinning of articular cartilage and small amounts of fluid.

DIFFERENTIAL DIAGNOSIS

Generally, one must consider conditions with articular calcifications and ossifications, soft tissue masses and loose body formation.

Osteoarthritis. This frequently involves the knee joint and may be associated with osteophytes which break off and become loose bodies. The latter, however, are generally fewer in number than in synovial osteochondromatosis and are larger, coarser and often have lucent centers. These coarse bodies are commonly mistaken as indicative of synovial osteochondromatosis but synovial biopsy reveals no metaplasia or thickening. Osteoarthritis secondary to true synovial osteochondromatosis can occur but is mild and will retain the fine, coalescent stippling of the primary entity. Osteoarthritis which is not related to trauma, infection or anomalies usually occurs in an older age group than synovial osteochondromatosis. Soft tissue masses are not seen in osteoarthritis.

Pigmented Villonodular Synovitis. This presents with nodular articular soft tissue masses but calcification is almost always absent.[6] Bone erosion resembling our Case 1 can be seen but occurs in joints with small articular spaces such as the elbow and wrist rather than the knee. A dark brown or serosanguineous fluid is often obtained when the joint is aspirated.

Synovial Sarcoma. This highly malignant tumor usually originates from paraarticular soft tissues rather than from within the joint. Differentiation from synovial osteochondromatosis may be difficult, especially if bone erosion and destruction cannot be distinguished. However, a largely extra-articular mass with adjacent bone destruction rather than erosion is suggestive. Calcification is not infrequent in synovial sarcoma and its presence is therefore not helpful in differentiation.

Osteochondritis Dissecans. The characteristic cartilage covered button of necrotic bone may float free within the joint or even become completely absorbed but a

tell-tale localized depression in the articular surface will remain. Loose bodies from this entity are usually few in number and coarse as compared to the numerous, fine bodies in synovial osteochondromatosis.

Neuropathic Arthropathy. Knee involvement in tabes and less frequently in diabetes will give extensive, coarse intra-articular calcification. However, extensive sclerosis is present in the adjacent metaphyses together with hydroarthrosis and severe proliferative changes in the joint. When joints other than the knee are involved with neuropathic arthropathy, differentiation is even easier.

Hemangioma. This occurs primarily in the synovium and capsule of the knee joint and probably results from local congenital vascular malformations. An intra-articular or para-articular water density mass is seen without bone erosion. Only occasionally can phleboliths be recognized and then there is usually angiomatous involvement and calcification of para-articular musculature.

Popliteal Artery Aneurysm. This occurs immediately posterior to the joint or somewhat higher. Expansion of the calcified vessel is evident. Dispersal of calcified flecks adjacent to the expansion has been said to indicate aneurysmal rupture[1] but angiography is necessary for definitive diagnosis.

Chondrosarcoma. The confluent fine calcifications seen in synovial osteochondromatosis may rarely resemble chondrosarcoma. However, the latter is usually para-articular and will often be seen together with exostoses or enchondromata and bone destruction.

Miscellaneous. For completeness the rare lipomata and fibromata should be mentioned. These contain no calcium and do not affect adjacent bone.

Idiopathic Intra-articular Free Bodies. A group remains in which intra-articular loose bodies of varying degrees of calcification and coarseness are seen in the absence of any of the above entities and without synovial metaplastic changes. The number of cases fitting this category obviously varies inversely with the degree of thoroughness and experience of the microscopist.

TREATMENT

Treatment requires the surgical removal of all accessible loose bodies to prevent mechanical articular damage. In addition, synovectomy is performed to exclude a potential source of new intra-articular masses. Surgery is generally successful although the results are modified by the degree of secondary osteoarthritis present preoperatively. Surgically inaccessible calcified free bodies, usually in bursae, may be seen on the postoperative roentgenogram but these have been reported to decrease or even disappear. Radiation therapy is not generally advocated.

CONCLUSION

Synovial osteochondromatosis is an uncommon joint disease characterized by metaplastic changes in the synovial sublining with resulting cartilage encrustation and free intra-articular bodies. Calcification and ossification occur.

Two cases are described.

Roentgenographically, calcification of a characteristic type is seen with intra-articular water density masses. Bone erosion may be seen.

Confusion with other entities can be avoided by an understanding of the clinical, pathologic and roentgen manifestations of synovial osteochondromatosis.

The differential diagnosis is discussed.

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IDIOPATHIC NONFAMILIAL ACRO-OSTEOLYSIS ASSOCIATED WITH OTHER BONE ABNORMALITIES*

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ACRO-OSTEOLYSIS refers to lytic changes of the shafts of the distal phalanges, with preservation of the tufts and bases.[6] These manifestations may be familial or nonfamilial. Approximately 72 cases of familial acro-osteolysis have been reported.[2] The familial type is characterized by ulcerating skin lesions on the plantar surfaces of the feet, with secondary involvement of the distal phalanges. These bone changes appear first in the distal and middle phalanges and gradually progress proximally and may eventually involve the metatarsals.

The process is purely osteolytic, with no evidence of bone regeneration. Occasionally, osteomyelitis develops, due to secondary infection in the presence of an ulcer. The etiology is somewhat obscure, but the disease is hereditary, the inheritance being of the dominant type.[2] Some investigators believe that the disease is caused by a lesion of the peripheral nerve fibers.

Nonfamilial acro-osteolysis may be a secondary manifestation of a more generalized disease, or it may be idiopathic. The first case considered to be idiopathic was reported in 1950 by Harnasch.[3] The roentgen findings in Harnasch's case were bilateral defects in the distal phalanges of the fingers and toes with preservation of the terminal portions, defects of the metatarsal phalangeal joints and resorption of the alveolar processes of both upper and lower jaws.

Wieland[8] in 1952 described a similar case which he also considered idiopathic. A third case, the only one reported in the English literature, was presented by Greenberg and Street[1] in 1957, who suggested that the condition was both idiopathic and nonfamilial.

Since Harnasch's original article, 5 other cases[4,5,7] with similar roentgenographic findings were reported, and these cases were not considered to be idiopathic. The diagnoses were established as absorption deficiency, hunger osteopathy, endocrine deficiency, and hyperparathyroidism, both primary and secondary.

It is our purpose in reporting a case, which we consider nonfamilial and idiopathic, to (1) support the opinions of Harnasch[3] and Greenberg and Street[1] that this does represent a new disease entity and to (2) suggest that this disease is probably a more generalized osseous dysplasia in which acro-osteolysis is the major manifestation.

REPORT OF A CASE

T.W., a twenty-three year old white female, was admitted to the hospital on February 16, 1959 at which time she was approximately thirty-six weeks pregnant. Admission was advised because the patient exhibited pitting edema of both legs and had a moderate anemia. Her pregnancy otherwise had been uncomplicated. Past history revealed that during her first delivery she required a blood transfusion because of anemia.

Physical examination revealed an unusual bony ridge in the occipital area of the skull above the protuberance. Several teeth were noted to be absent, including the mandibular and maxillary incisors. Most of the remaining teeth were very mobile. There was a peculiar shortening and blunting of the distal portions of some of the fingers (Fig. 1*A*) and toes. The nails were intact, with low longitudinal ridges. The patient stated that she had had trouble with her teeth since birth, and also that the

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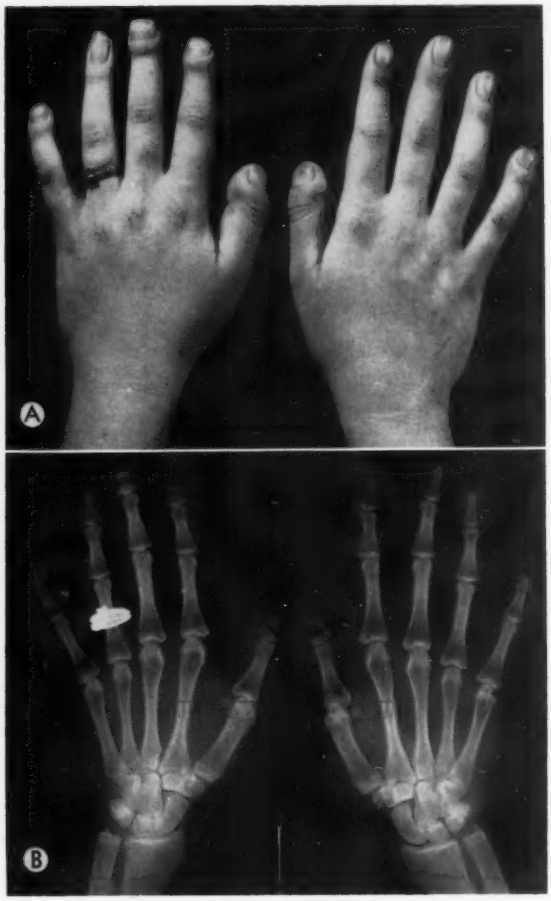

FIG. 1. (*A*) The hands show distal shortening and blunting of several of the digits. The nails are intact, with low horizontal ridges. (*B*) Several of the distal phalanges are shortened, with absorption of the mid-portions and sparing of the tufts and bases. The head of each fifth metacarpal shows a single cystic defect.

peculiar appearance of her fingers and toes was present as far back as she could remember, and had not changed. There was no evidence of any skin manifestations, including ulcerations, or of any neurologic changes. Examination of the chest revealed a Grade II systolic murmur over the precordium, but there was no evidence of enlargement of the heart clinically. There was nothing to suggest any endocrine disturbances or absorption deficiencies.

Laboratory data revealed a hypochromic anemia which responded to blood transfusions. All of the blood chemistries, including calcium, phosphorus and alkaline phosphatase, were within normal limits.

On March 15, 1959 the patient delivered a viable female. Delivery was spontaneous and uncomplicated. The infant was noted to be normal both clinically and roentgenographically. The patient's first child was at this time three years old, in good health and had no obvious congenital abnormalities. There was no history or any evidence of any congenital defects in any member of the patient's family.

Roentgenograms of the patient's hands and feet made following delivery revealed osteolytic defects involving the mid-portions of some of the distal phalanges of the fingers and toes (Fig. 1*B*, 2, 3 and 4). There were several small cystic defects in the proximal phalanges of both great toes. There was a linear radiolucent defect involving the proximal diaphysis of both the left fifth metatarsal and the right fourth metatarsal (Fig. 4). These changes resembled an ununited fracture. However, there was no evidence of any callus formation or bone sclerosis. Roentgenograms of the long bones (Fig. 5, *A* and *B*) revealed lack of tubulation, with widening of both the proximal and distal portions of the diaphyses.

The skull (Fig. 6*A*) showed an abnormal contour with a bulging posteriorly at the inion, giving the skull a dolichocephalic shape. All of the sutures were ununited and there was a persistent metopic suture. The frontal sinuses were absent. There were multiple wormian bones along all the suture lines, giving the skull a mosaic appearance. The sella turcica was normal. There was prominent bone resorption of

FIG. 2. Magnification of the phalanges of the thumbs demonstrating the characteristic defects of the distal phalanges.

Fig. 3. Multiple defects of the phalanges, including cystic changes in the proximal phalanges of the great toes, absorption of the distal articular ends of the proximal phalanges of both fifth digits, and absorption of the tufts of several distal phalanges.

the alveolar processes of the maxillae and the mandible (Fig. 6*B*). Several teeth were absent and most of the remaining were loosely held in their sockets. There was also partial resorption of the roots of the remaining teeth, with absence of the lamina dura. Roentgenograms of the chest, spine and pelvis were normal.

Fig. 4. Defects of the proximal diaphyses of both the left fifth metacarpal and the right fourth metacarpal, resembling ununited fractures. There is no evidence of callus formation or bone sclerosis.

DISCUSSION

The authors feel that this case presents findings identical to those reported by Harnasch,[3] Wieland,[8] and Greenberg and Street,[1] and supports the impression that the condition is a nonfamilial and idiopathic entity. In addition, the present case reveals other bone abnormalities not previously described; namely, multiple wormian bones of the skull and lack of tubulation of the long bones. In view of these additional findings, the authors feel that so-called idiopathic nonfamilial acro-osteolysis represents only one manifestation of a more generalized congenital osseous dysplasia.

SUMMARY

A case of idiopathic nonfamilial acro-osteolysis with additional associated ab-

Fig. 5. (*A* and *B*) The long bones demonstrate lack of tubulation, with widening of the proximal and distal portions of the diaphyses.

Fig. 6. (*A*) The skull exhibits an abnormal contour with a bulging posteriorly at the inion, giving the skull a dolichocephalic shape. All of the sutures are ununited and there are multiple wormian bones along all the suture lines, giving a mosaic appearance. (*B*) The mandible and maxilla show prominent absorption of the alveolar processes. Several teeth are absent and most of the remaining ones are held loosely in their sockets. The roots show partial resorption and the lamina dura is absent.

normalities is reported, representing the fourth case of its kind in the world literature.

The reported case is unique because of the additional findings, indicating that nonfamilial acro-osteolysis may represent a generalized congenital osseous dysplasia.

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RETICULOHISTIOCYTOMA: A RARE DERMATOLOGIC DISEASE WITH ROENTGEN MANIFESTATIONS*

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RETICULOHISTIOCYTOMA is a grave condition of unknown etiology and difficult to diagnose, affecting the skin, bones and joints. Although distinctive roentgen features are present in half of the cases, no attention has been given to this subject in the radiologic literature. The roentgenographic recognition of reticulohistiocytoma is therefore of diagnostic value.

GENERAL CONSIDERATIONS

Reticulohistiocytoma is not a neoplastic process, but a definite entity of unknown origin recognized by dermatologists. Other names have been used to describe it, such as reticulohistiocytic granuloma, generalized giant cell histiocytosis or lipoid dermato-arthritis. The term "reticulohistiocytoma" seems adequate in describing the reticuloendothelial, histiocytic nature of the cells composing the skin nodules. These cutaneous lesions, which resemble xanthomas, appear first and are followed by arthritic symptoms of varying degrees of severity. Seventy per cent of the reported cases are associated with arthritis[3] which can progress to crippling destructive changes of multiple joints. Meanwhile the skin lesions involute, and in some patients the disease process resolves spontaneously.

It has been thought that this process represents a widespread granulomatous response to an unknown stimulus resulting in abnormal connective tissue metabolism.[2] Thyroid dysfunction has been linked to this condition after observing that in some patients the clinical picture improved after thyroidectomy. These and other implications are conjectural and leave the pathogenesis of this disease still unknown. Although high blood cholesterol has been reported in some patients, the blood chemistries reveal no typical abnormal findings. The blood cell count, urinalysis and liver function tests are also normal. The final diagnosis is made by biopsy of the cutaneous lesions. Typical giant, multinucleated cells are evident in the biopsy sections of the skin nodules and synovium;[4] nevertheless, they can be mistaken for cells found in histiocytoma, myoblastoma and ganglioneuroma,[1] although the latter can be differentiated by the presence of Nissl's granules in the thionine stain. Sections stained with Sudan IV fail to reveal any considerable deposition of lipid in these cells, thus ruling out xanthomatosis.

ROENTGENOLOGIC FEATURES

In the typical cases, the articular surfaces of the bones are destroyed rapidly and cartilage destruction proceeds slowly with extensive involvement of the distal interphalangeal joints of the hands and feet particularly.[5] In these joints, the early changes appear as small areas of paraarticular destruction with some loss of the articular space and minimal osteoporosis of the adjacent structures (Fig. 1, *A* and *B*). At this stage they may resemble rheumatoid, gouty or psoriatic arthritis. Later there is a progressive demineralization and absorption of the articulating ends of the long bones, especially those of the hands and feet (Fig. 2, *A* and *B*). Other joints are involved such as the posterior articulations of the dorsolumbar spine, hips, shoulders, wrists, and connective tissue attachments of femoral trochanters and at the distal ends of clavicles (Fig. 3, *A*, *B*, *C* and *D*). The flat bones are not involved except for the glenoid process of the scapula and occasionally the ribs. The changes progress to

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FIG. 1. (*A*) Roentgenograms of the hands reveal narrowing of the interphalangeal spaces and para-articular punched-out areas of bone destruction, more predominant in the distal joints but also seen in the metacarpals. (*B*) Similar changes are seen in the feet, particularly at the head of the first metatarsal. The roentgen impression was that of gouty arthritis but the diagnosis of reticulohistiocytoma was made by biopsy. (Case reported by Dr. M. R. Caro.)

a complete destruction of the articular ends of the long bones of hands and feet with telescoping of the digits due to total disappearance of the interphalangeal joints. The phalangeal shafts then appear as small fragments of bone with pointed ends and sharp borders (Fig. 4, *A*, *B* and *C*).

Not all patients show these multilating

Fig. 2. More severe changes were found in the hands and feet of this forty year old woman who had numerous skin nodules and arthritic symptoms of one year's duration. A diagnosis of reticulohistiocytoma was made by biopsy of the lesions. (*A*) Note the severe destruction of the articulating ends of the phalanges and heads of the metacarpals. Some irregular areas of bone destruction are also present in the carpal bones and articular ends of the radius and ulna. (*B*) Roentgenograms of the feet show total loss of the interphalangeal spaces with destruction of the adjacent articular ends and osteoporosis of the underlying regions.

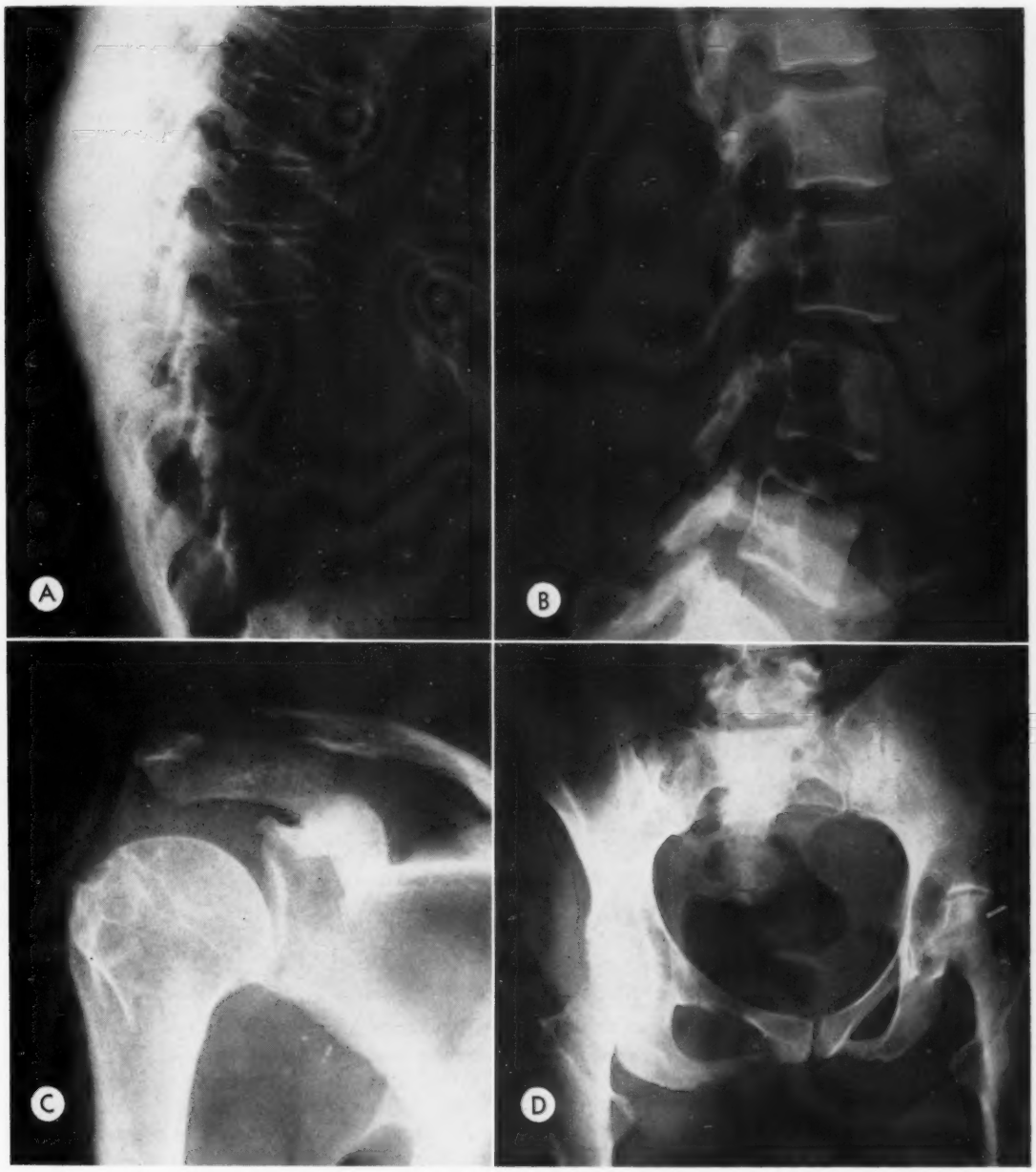

Fig. 3. Same patient as in Figure 2. (*A* and *B*) Involvement of the thoracic and lumbar spine is seen with erosion and destruction of the articular facets and processes of the posterior vertebral articulations. (*C*) Similar changes are noted in both shoulders which show irregular areas of destruction at the distal ends of the clavicles. Punched-out areas are seen at the glenoid process and humeral head. (*D*) Acetabular and femoral head changes are seen in this patient as areas of radiolucency, representing bone destruction at these levels.

changes; a spontaneous involution of the process can occur.

SUMMARY

Reticulohistiocytoma is a non-neoplastic, dermatologic disease of unknown origin producing skin nodules and arthritic symptoms. Patients with this condition are in good health until the sudden appearance of nontender, painless lumps in the skin of the extremities, trunk, face, and in the oral mucosa. Some nodules tend to disappear or recur later associated with stiffness of the fingers, elbows and sometimes the knees.

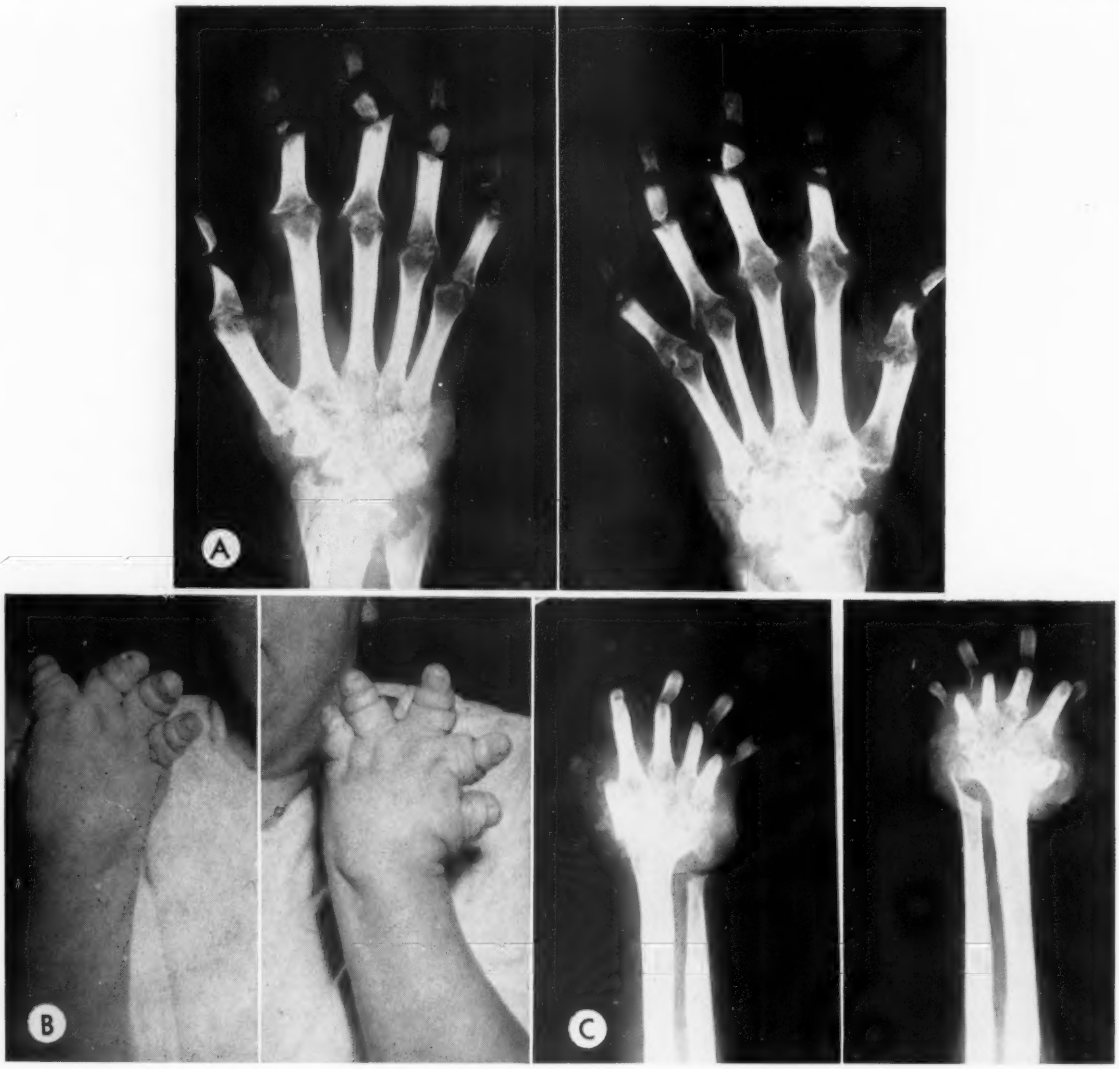

Fig. 4. (*A*) Roentgenograms of the hands of a thirty year old patient with reticulohistiocytoma of four years' duration. Destruction of the articulating ends of the phalanges and metacarpals is noted. There is erosion of the distal ends of the radius and ulna, and of several carpal bones. (*B*) Telescoping of the digits is seen in the same patient seven years after the onset of her illness. (*C*) Severe absorption of the phalanges and disappearance of the interphalangeal joints has occurred. The carpal bones as well as the articular ends of the radius and ulna are partially absorbed. (By permission of *A.M.A. Arch. Dermat.*—Johnson and Tilden—March, 1957.)

The roentgen features vary depending on the severity of the disease and are sometimes mistaken for changes due to rheumatoid, psoriatic or gouty arthritis. More typical changes such as progressive demineralization, absorption and destruction of the articular ends of the long bones of the hands and feet are seen in advanced cases. Other joints can be involved in the same peculiar fashion, with absorption of the bone in contact with the synovium. The posterior vertebral joints and hips are affected in some patients. The disease may involute spontaneously or progress to a severe, crippling state.

It is not possible to establish a diagnosis

clinically, since the skin nodules resemble those of other diseases. The biopsy sections show typical cells.

It is important that the radiologist be aware of this condition to assist in the diagnosis of this serious, rare and seldom recognized disease.

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POLYOSTOTIC BONE SARCOMA ASSOCIATED WITH OSTEITIS DEFORMANS

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THE occurrence of bone sarcoma as a complication of Paget's disease of bone is a well known fact. Sir James Paget[9] noted the combination of osteogenic sarcoma and osteitis deformans in at least 5 of the 23 patients he described during the nineteenth century. In 1946 Summey and Pressly[13] reported on 76 instances of sarcoma complicating osteitis deformans culled from the literature. The roentgen diagnostic features of sarcomatous degeneration in Paget's disease were thoroughly described by Sherman and Soong.[11] The frequency of this association is reported as varying from 0.9–30 per cent[10] with the majority of authors reporting an incidence of from 7–11 per cent. Assessment of this incidence is difficult because of a large number of asymptomatic cases of Paget's disease that are not diagnosed. However, the frequent occurrence of polyostotic sarcomas in Paget's disease has not been emphasized in the radiologic literature.

POLYOSTOTIC OSTEOGENIC SARCOMA

Osteogenic sarcoma originating in apparently normal bone almost always starts in a single bone and usually is rapidly fatal after metastasizing to the lungs and other nonosseous structures via the blood stream. There are a few reports of osteogenic sarcoma occurring at several osseous sites in apparently normal bone.[1,12] The patients so afflicted have been children or young adults. Because the sarcomas appear to arise simultaneously at their various osseous sites, it has been suggested that these malignant tumors are multicentric in origin. Primary polyostotic osteogenic sarcoma is very rare.

POLYOSTOTIC OSTEOGENIC SARCOMA IN PAGET'S DISEASE

Osteogenic sarcoma complicating osteitis deformans is not uncommonly found in several osseous sites. Sherman and Soong[11] describe polyostotic sarcomas in 4 of 19 patients with Paget's disease. Twenty-six of the 76 individuals reviewed by Summey and Pressly[13] showed multiple bone involvement with sarcoma. Eighteen patients showed malignant involvement of two bones and in 8, more than two bones were sarcomatous. Both cases of osteogenic sarcoma and osteitis deformans reported by Davie and Cooke[4] showed malignant lesions in many bones. Porretta, Dahlin and Janes[10] reviewed the literature and reported on 128 cases of sarcoma developing in Paget's disease. Twenty-seven, or 21 per cent, were considered to be multiple. At the Presbyterian Hospital, 1 of 8 proved cases had multiple bone involvement. Others have reported similar findings. Kienböck and Selka[7] in 1935 stated that multiple osteogenic sarcomas arise only rarely at the site of Paget's disease, but a review of the reported cases casts doubt on this statement. These authors cite a forty-nine year old male with Paget's disease and multiple osseous and visceral sarcomatous metastases. Although their patient responded well to radiation therapy, most observers agree that none of the various therapeutic modalities now available have been of any value.

In most instances the multiple osteogenic sarcomas arise in bones already involved with osteitis deformans and therefore occur in association with polyostotic

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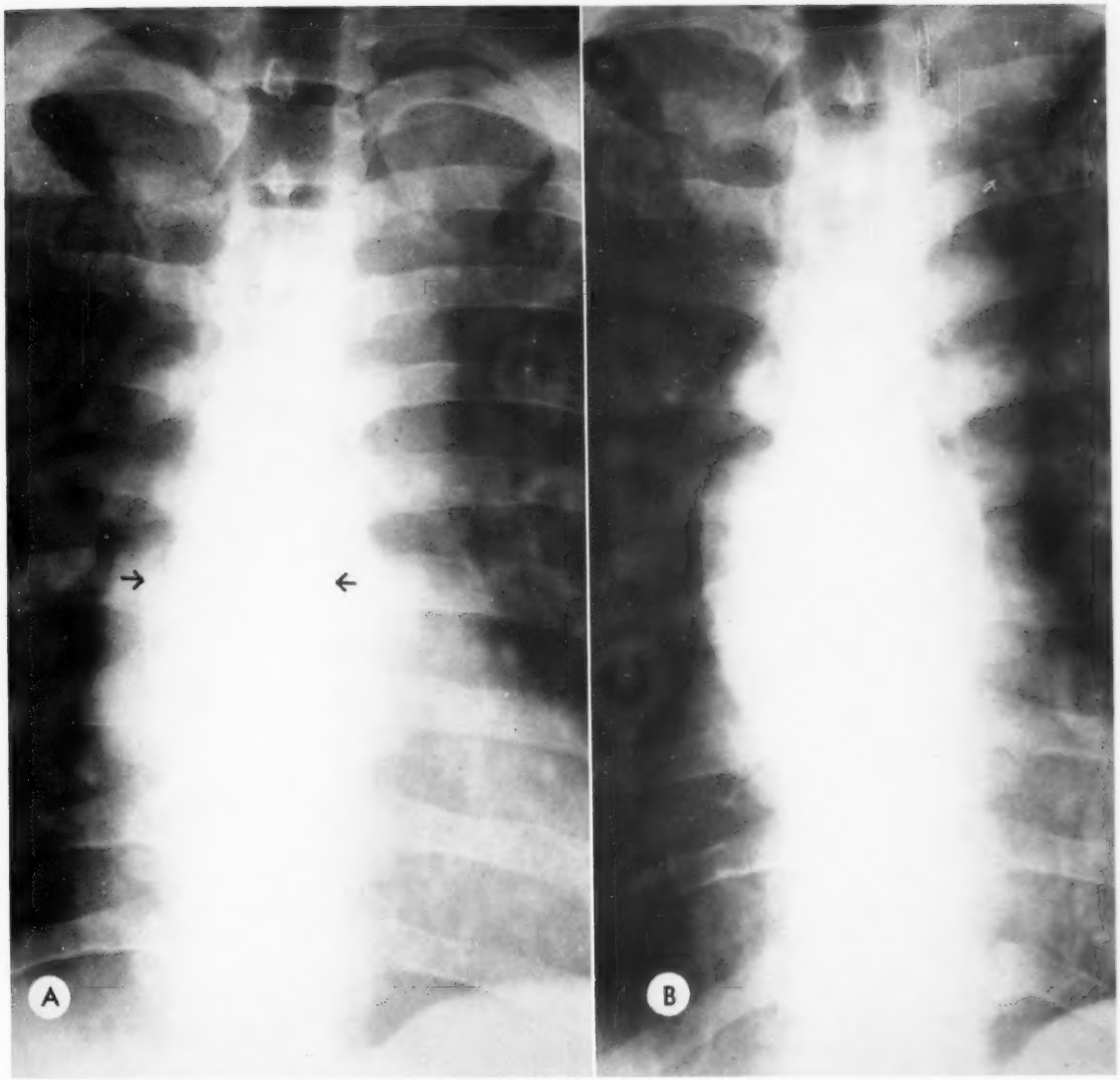

Fig. 1. Case 1. (*A*) Admission roentgenogram in 1949 showing osteoblastic changes involving T6, T7 and T8 with a partial collapse of the vertebral body of T7 (arrows). (*B*) Roentgenogram of thoracic spine in July, 1952 demonstrating destructive changes in the seventh vertebral body and a well demarcated paraspinal mass at the T6–T10 levels.

Paget's disease. Rarely do the bone sarcomas occur in normal surrounding bone.

REPORT OF CASES

Case 1. A forty-one year old white male paper cutter was first seen at the Presbyterian Hospital in 1949 with symptoms of compression of the mid-thoracic spinal cord. Spine roentgenograms revealed marked osteoblastic alteration involving T6, T7 and T8 with a partial collapse of the vertebral body of T7 (Fig. 1*A*). Similar bone changes were present in the lower lumbar spine and pelvis. Roentgenographic examination of the femur disclosed bowing anteriorly and marked expansion of the distal shaft with associated areas of radiolucency and irregular sclerosis (Fig. 2*A*). The diagnosis was Paget's disease of bone. The hemogram, urinalysis, sedimentation rate and serologic studies were all normal.

An emergency laminectomy provided relief of the mid-thoracic cord compression. The postoperative serum alkaline phosphatase was 31 Bodansky units and the serum phosphorus was

Fig. 2. Case 1. (*A*) Admission roentgenogram in 1949 demonstrating expansion of the distal shaft of the right femur with associated areas of radiolucency and irregular sclerosis. (*B*) Roentgenogram of right femur in 1952. There is an increase in the osteoblastic process and there are destructive bone changes in the metaphysis and epiphysis.

4.9 mg. per cent. Microscopic examination of the removed osseous specimen confirmed the diagnosis of Paget's disease.

The patient recovered and returned to work in December, 1950. He was then lost to follow-up.

In July, 1952, he returned with a painful right knee. Roentgenograms showed destructive changes in the distal end of the right femur (Fig. 2*B*). Roentgenograms of the thoracic spine disclosed areas of destruction in the body of T7 and the presence of a paraspinal mass (Fig. 1*B*). Accordingly, the proposed diagnosis was sarcomatous degeneration in Paget's disease.

Laboratory data included: normal hemogram, normal urinalysis, negative Mazzini test for syphilis, serum alkaline phosphatase 119.6 Bodansky units, serum acid phosphatase 6.8 Gutman units (slight increase thought related to elevation of the alkaline phosphatase), urine negative for Bence-Jones protein, total serum protein 7.9 gm. per cent with a 1.5 A/G ratio, serum nonprotein nitrogen 40 mg. per cent, serum calcium 9.3 mg. per cent and serum phosphorus 4.4 mg. per cent.

Osteogenic sarcoma was present in a biopsy obtained from the distal end of the right femur. No therapeutic surgery was attempted and the patient was referred for radiation therapy.

When first seen in the therapy department, he complained of a severe headache and a mass was palpable in the right frontal area. This represented one of many metastases to the cranial bones.

The cranial lesion received 1,270 r tissue dose in four days using an 8 cm. circular cone. The right knee was treated with two opposing 15×20 cm. ports and a 2,000 r tissue dose was delivered in nine days. The tumor dose of the left hemipelvis was 1,500 r in eleven days. The physical factors were: 250 kv. peak; 1 mm. Cu half value layer, and 50 cm. target skin distance.

The radiation therapy was concluded on August 29, 1952, at which time the patient

noted partial relief of pain in all of the treated sites.

In October of the same year, he returned with pathologic fractures of the left humerus and femur. A biopsy of the left femur revealed osteogenic sarcoma and Paget's disease.

Rush nails were inserted into the recent fractures but he fared poorly following surgery and expired on October 23, 1952 (thirty-three months after the initial diagnosis of Paget's disease and three months subsequent to the detection of sarcomatous degeneration).

A postmortem examination revealed widespread Paget's disease with sarcomatous changes involving the spine (Fig. 3), femora, left humerus, left clavicle, calvarium, ribs, pelvis and probably right fifth metacarpal. Sarcoma was not found in bone free of osteitis deformans. There were metastases to lungs, pancreas, pleura, diaphragm, dura and adjacent cerebral cortex.

In additon there was hypertrophy of the left ventricle and slight generalized cardiac dilatation. The cause of death was cardiac failure

Fig. 3. Case I. Postmortem roentgenograms of the spine demonstrating disseminated Paget's disease with areas of neoplastic bone destruction at several levels. Sarcomatous changes were confined to bones involved by osteitis deformans.

Fig. 4. Case II. Admission roentgenogram in August, 1954, showing characteristic findings of osteitis deformans in pelvis and lumbar spine.

possibly related to the increased blood flow in disseminated Paget's disease.

Case II. E.J., a fifty-three year old white male was well until March, 1954 when he developed perianal pain. This persisted, later accompanied by paresthesias and loss of sensation associated with defecation and micturition.

In August, 1954, he was admitted to the Syracuse Memorial Hospital.

The salient physical findings included hypesthesia over the buttocks, perineum and upper posterior thighs. The anal, abdominal and ankle reflexes were absent bilaterally and dorsiflexion of the left foot was diminished.

The serum alkaline phosphatase was elevated to 28.1 Bodansky units and roentgenograms exhibited Paget's disease in the right ilium, both ischia and the second lumbar vertebral body (Fig. 4). The hemogram, urinalysis, nonprotein nitrogen, blood sugar, serum calcium, serum phosphorus, serum acid phosphatase and Wassermann test were all normal.

On August 3, 1954 a lumbar puncture yielded clear fluid under normal pressure. The

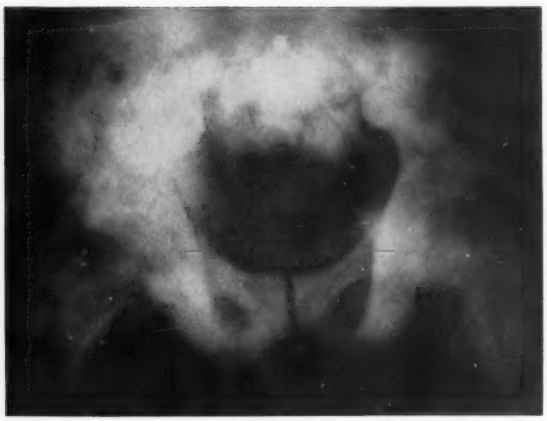

Fig. 5. Case II. Roentgenogram made in October, 1954 when there was obvious progression of the osteolytic and osteoblastic changes in the pelvis. A biopsy of the right iliac crest revealed osteogenic sarcoma. There is a small amount of opaque material in the subarachnoid space.

total protein was 102 mg. per cent, sugar 81 mg. per cent and the chlorides 125 mEq. L.

On August 9, 1954 a lumbar myelogram was performed using 9 cc. of pantopaque. The roentgenologic report was as follows: "Examination of the lumbosacral spine and pelvis shows thickening of the cortex involving the entire pelvis as well as coarsening of the trabecular pattern. The appearance of the pelvis is entirely characteristic of Paget's disease. In addition there is increased density of the body of the second lumbar vertebra. In view of the pelvic changes it is probable that the sclerotic alteration in the lumbar spine denotes Paget's disease. However, there is irregularity of the inferior margin of the second lumbar vertebral body suggesting the possibility of true destruction secondary to a malignant neoplasm." The myelogram did reveal a rather large extradural defect on the left side opposite the altered vertebra.

A decompression laminectomy was carried out on August 19, 1954. At the time of surgery the spinous processes of L1, L2 and L3 appeared abnormally soft. The neural arch of L2 was thicker than usual and the epidural fat was almost absent. There was narrowing of the neural canal at this level. The removed osseous fragments disclosed microscopic features of Paget's disease.

The early postoperative course was favorable; the saddle type anesthesia disappeared and there was a return of good sphincter tone.

The final hospital admission was in October, 1954 when fecal incontinence recurred and he was unable to void. Additional complaints were pain at the "base of the spine" radiating down both lower extremities and numbness of the right leg.

Laboratory data included: a faint trace of albuminuria, hemoglobin 12 gm., white blood cell count 12,800, neutrophils 85 per cent, lymphocytes 14 per cent, eosinophils 1 per cent, nonprotein nitrogen 38 mg. per cent, serum acid phosphatase 0.4 King-Armstrong units, serum alkaline phosphatase 14.7–29.1 Bodansky units, and total serum protein 7 gm. per cent.

Roentgenographic study of the pelvis (Fig. 5) demonstrated rapid increase in progression of the osteolytic and osteoblastic changes.

A biopsy of the right iliac crest was obtained and the pathologist's report was osteogenic sarcoma arising in Paget's disease.

Palliative orthovoltage roentgen therapy was administered to the pelvis in an attempt to relieve pain. He received 2,000 r tissue dose in two weeks through an anterior 10×15 cm. port.

A refractory anemia developed and a rapid downhill course ended in death on January 28, 1955 (ten months after the diagnosis of Paget's disease with probable superimposed sarcoma and approximately one month after the presence of sarcoma was proved). Permission for postmortem examination was not obtained.

DISCUSSION

Osteitis deformans may occur in young adults but it is rarely seen under forty years of age. Porretta, Dahlin and Janes[10] in their analysis of 104 cases of sarcoma complicating Paget's disease found the average age to be 59.6 years. This, of course, contrasts sharply with primary osteogenic sarcoma in which the average age of onset is between ten and thirty years.

In contrast to primary osteogenic sarcoma, the sarcomas complicating osteitis deformans exhibit the interesting characteristic of often simultaneously affecting multiple bones of the same individual. This was demonstrated in the 2 patients cited above.

In the first patient there were multiple bone and visceral neoplasms and in the second the changes were confined to the osseous system. As far as could be determined there were no sarcomatous changes in bones not involved by osteitis deformans.

The above case histories demonstrate the aggressive nature of these sarcomas. There have been no known cures. Surgical amputation, when possible, would seem to be the preferable treatment. Radiation therapy has been used to control pain in the terminal stages.

The cause of both osteitis deformans and osteogenic sarcoma is unknown. It is therefore hazardous to speculate whether the polyostotic malignant lesions are multicentric due to some humoral or other mechanism or whether the polyostotic involvement represents actual metastasis. However, several facts do support the multicentric origin theory. As was demonstrated in the 2 cases reported here and appears to be true in most of the case histories published elsewhere, when multiple sarcomas develop they usually occur in bone already involved with Paget's disease and tend not to develop in apparently previously normal bone. Not infrequently many bones are involved extensively with sarcoma and there are few or no visceral metastases present. On the other hand, the primary osteogenic sarcoma usually metastasizes to lungs and other nonosseous structures. The usual primary osteogenic sarcoma is not polyostotic.

These facts, of course, do not establish the multicentric hypothesis. A few instances have been described in which multicentric osteogenic sarcoma occurred in a patient with Paget's disease and in which apparently normal bone was involved with sarcoma.[7] In addition, there are examples in which extensive visceral metastases were present and in which one could not deny the possibility that bone metastases have actually occurred.

Obviously, discovery of the basic mechanism involved in the development of Paget's disease as well as of the factors that cause malignant degeneration in pagetoid bone must be made before this question can be answered.

CONCLUSION

1. Two cases of polyostotic Paget's disease in which osteogenic sarcoma developed in multiple bones are reported.

2. The usual primary osteogenic sarcoma is not polyostotic in origin and bone metastases are extremely rare.

3. Multiple bone sarcomas are not uncommon when osteitis deformans is complicated by osteogenic sarcoma.

4. Whether the multiple bone sarcomas actually represent multicentric origin of sarcoma in Paget's disease or are the result of metastases cannot be stated at present.

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SKELETAL METASTASES FROM MALIGNANT TESTICULAR TUMORS*

A REPORT OF 10 CASES WITH OSTEOLYTIC AND OSTEOBLASTIC CHANGES

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SECONDARY skeletal involvement in patients with malignant testicular tumors has been documented rather infrequently. It is our purpose to report 10 such cases from a group of 167 patients with neoplasms of the testis, seen or treated in the Radiation Clinic of the Veterans Administration Hospital from 1949 to 1957. The ages of the subjects to be reported ranged from twenty-five to forty-two years. Their primary lesions included 5 seminomas, 4 embryonal carcinomas, and 1 teratocarcinoma. Seven patients exhibited roentgenographic evidence of skeletal involvement during life, with histologic confirmation by surgical biopsy and/or autopsy. Of these, roentgenograms revealed osteolytic lesions in 5 patients, while multiple osteoblastic foci were disclosed in 2 individuals. In the remaining 3 cases, metastatic bone lesions were found only at postmortem examination.

In most of these patients, multiple osseous lesions either appeared at different intervals or simultaneously. The skull was involved in 1 instance; the vertebrae in 7; the ribs in 4; the femora in 3; the humerus in 1; and the coracoid process of the scapula in 1 instance.

REPORT OF ILLUSTRATIVE CASES

CASE 1. A.R., a forty year old male, was admitted to the hospital on March 28, 1957, with progressive inability to walk of five days' duration. Eighteen months prior to admission, the patient had an orchiectomy for seminoma of the right testicle at another hospital and received postoperative irradiation to the groin and abdomen. In November, 1956, he had a sudden onset of right hemiplegia and eight days later experienced a seizure with twitching of the right face. For this problem, he received irradiation to the skull.

Myelography on admission to our institution disclosed a complete block at the level of the fifth thoracic vertebra. The latter appeared dense on the plain roentgenogram (Fig. 1*B*). Laminectomy and excision of an epidural tumor was accomplished on March 29, 1957 and the histology was reported as metastatic seminoma. The patient received postoperative irradiation through a single port to the operative site. A tumor dose of 2,250 r was administered in eighteen days, employing a 1,000 kv. apparatus with a half-value layer of 3.9 mm. lead. Further roentgen therapy to the retroperitoneal lymph node areas was given through five ports and a tumor dose of 2,100 r was administered from May 16, 1957 to June 5, 1957. At the completion of this course of treatment, he was given rehabilitation therapy.

In August, 1957, the patient developed pain in both lower extremities. Roentgenograms showed osteoblastic changes in the neck of the left femur (Fig. 1*C*), in the right lower femur (Fig. 1*D*), osteolytic and osteoblastic changes in the left lower femur (Fig. 1*E*), as well as osteolytic changes in the proximal right tibia. Biopsy from the left lower femur disclosed metastatic seminoma. There were no pulmonary metastases but the seventh and eighth ribs on the right side and the seventh rib on the left side showed osteoblastic changes (Fig. 1*A*). In October, 1957, the patient noted a firm mass, 6 cm. in diameter, intimately related to the external surface of the left mandible at its angle. In November, 1957, osteoblastic and osteolytic areas were found in both humeri and in the coracoid process of the right scapula (Fig. 2, *A* and *B*). All of these bone lesions except those of the ribs were subsequently irradiated to a tumor dose level of 2,000–3,000 r because of severe pain. The lesions were quite radiosensitive and the pain promptly disappeared. The patient was discharged to his home immediately after the course of irradiation.

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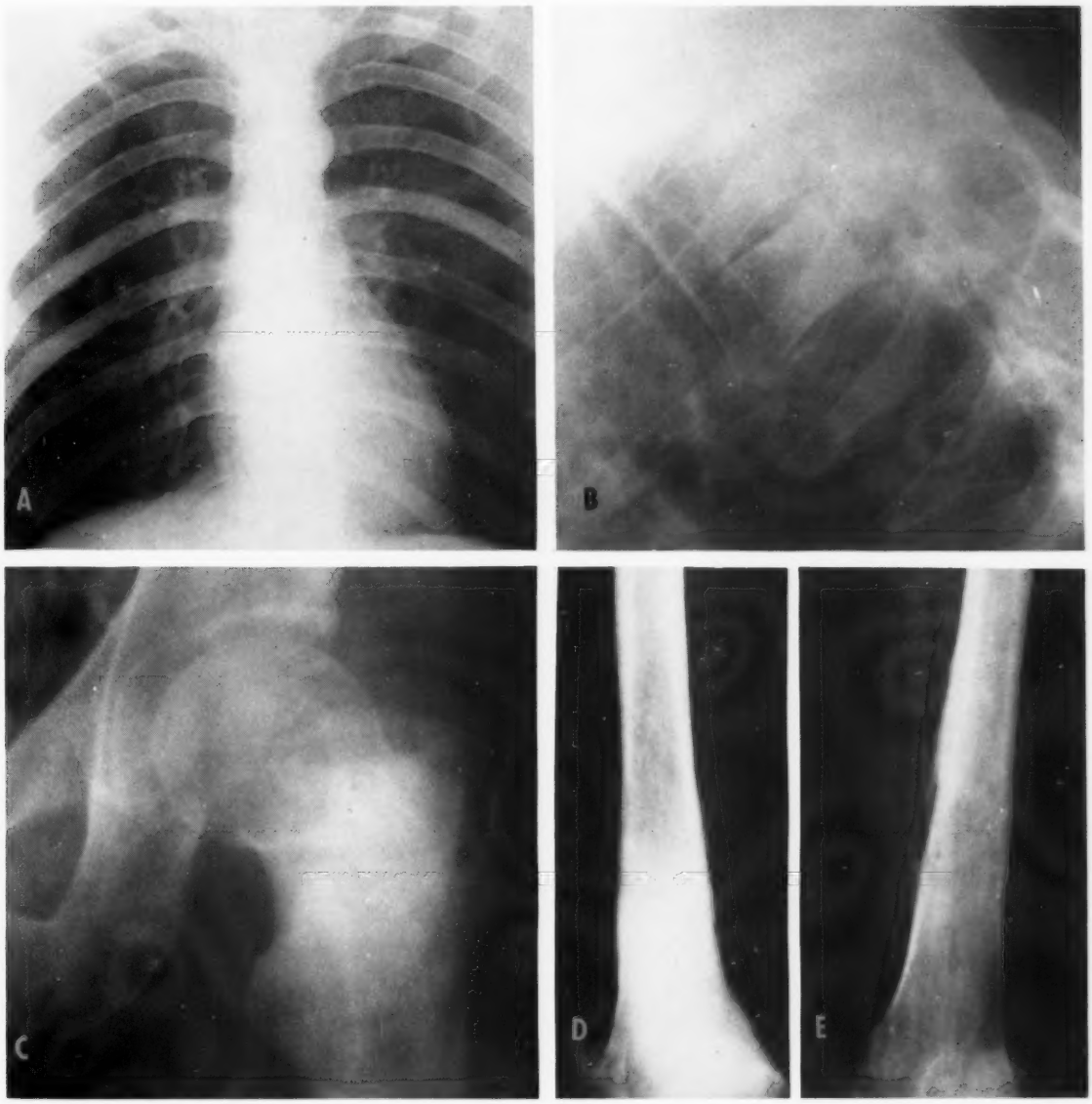

Fig. 1. Case 1. There is evidence of osteoblastic metastases to: (*A*) the right seventh and eighth ribs and left seventh rib; (*B*) the body of the fifth vertebra; (*C*) the neck of the left femur; and (*D*) the lower half of the shaft of the right femur. (*E*) The left femur shows both osteoblastic and osteolytic involvement.

Case II. W.S., a thirty-nine year old white male, had had an orchiectomy for seminoma of the left testicle in September, 1954, with subsequent retroperitoneal lymph node dissection. These lymph nodes were positive for seminoma on histologic examination. Irradiation of the abdomen was accomplished through five ports. A tumor dose of 2,000 r was administered to the upper and lower abdomen, employing a 1,000 kv. apparatus.

In February, 1956, the patient developed pain in the right hip radiating down the posterior thigh. Roentgenograms showed osteoblastic areas consistent with metastatic neoplasm, involving the neck of the right femur (Fig. 3*C*) and the right eighth rib anteriorly (Fig. 3*A*). These areas were irradiated from February 26, 1956 to March 9, 1956 to a tumor dose level of 1,700 r to the right hip and 1,750 r to the right eighth rib, with alleviation of bone pain.

In March, 1956, the patient noted a right postauricular swelling with marked pain which responded to 900 r (tumor dose) administered in six days. In September, 1957, a tender mass (4 cm. in diameter) was seen on the right lower chest wall, attached to the seventh and eighth ribs on their lateral aspects. Roentgenograms

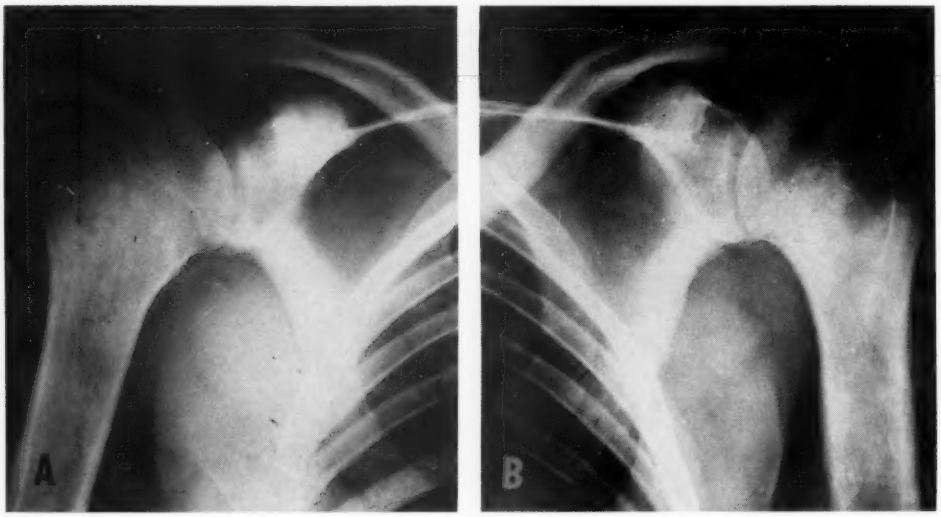

Fig. 2. Case I. (*A*) The coracoid process of the right scapula is very dense. The humerus shows osteolytic and osteoblastic changes. (*B*) The coracoid process of the left scapula appears normal. Note metastatic deposits in the humerus.

revealed osteoblastic changes involving these ribs. Both mass and pain disappeared after a tumor dose of 1,940 r given from September 10, 1957 to October 3, 1957. However, the osteoblastic changes persisted. The patient was discharged and at present is working as a janitor.

Case III. P.J.M., a twenty-six year old white male, complained of severe headaches, pain and weakness of both lower extremities of three to

Fig. 3. Case II. (*A*) Osteoblastic involvement of the right eighth rib as compared with (*B*) the normal ribs on the left side. (*C*) Osteoblastic involvement of the neck of the right femur as compared with (*D*) the normal neck of the left femur.

TABLE I

CASE RECORDS

Patient	Diagnosis	Age at admission (yr.)	Survival Time after Onset	Treatment	Metastases during Life	Skeletal Roentgenographic Findings	Autopsy Findings
W.R.G.	seminoma, rt.	25	36 mo.	orchiectomy, Sept., 1951 and postoperative irradiation	rt. cervical lymph nodes and lt. femur	osteolytic metastases	no autopsy
J.J.B.	teratocarcinoma, rt.	39	25 mo.	orchiectomy, April, 1951, retroperitoneal lymph node dissection, May, 1951 and irradiation, Aug., 1952	retroperitoneal lymph nodes	negative	widespread involvement, including temporal skull, D2, D5 and D7 vertebrae
C.H.	embryonal carcinoma, lt.	28	20 mo.	orchiectomy, Sept., 1949, and postoperative irradiation	lungs, retroperitoneal lymph nodes and supraclavicular lymph nodes	negative	widespread involvement, including L3, L4 and L5 vertebrae
J.J.M.	seminoma, lt.	42	22 mo.	orchiectomy, Oct., 1948, and postoperative irradiation	Lungs and L3 vertebra	osteolytic metastases	widespread metastases, including skeleton
J.F.	embryonal carcinoma, rt.	28	22 mo.	orchiectomy, Jan., 1951, and irradiation	cervical lymph nodes, D4 and D5 vertebrae and rt. third and fourth ribs	osteolytic metastases	widespread metastases, including skeleton
A.J.C.	embryonal carcinoma, rt.	34	14 mo.	orchiectomy, April, 1953, and laminectomy, Oct., 1953	retroperitoneal lymph nodes and D12 vertebra	osteolytic metastases	widespread involvement, including D12, L1 and L2 vertebrae
H.D.C.	embryonal carcinoma, lt.	31	8 mo.	orchiectomy, Jan., 1953, and postoperative irradiation	lt. chest wall, lungs and liver	negative	widespread metastases, including ribs
P.J.M.	seminoma, rt.	26	6 mo.	radiation therapy to retroperitoneal mass	lungs, retroperitoneal mass and L2 vertebra	osteolytic metastases	widespread metastatic embryonal carcinoma and nodule in rt. testicle reported as seminoma
A.R.	seminoma, rt.	40	living with disease	orchiectomy, Sept., 1955, and postoperative irradiation	D5 vertebra, ribs, femora and humeri	osteolytic and osteoblastic metastases	alive
W.F.S.	seminoma, lt.	39	living with probable disease	orchiectomy, Sept., 1954, and postoperative irradiation	retroperitoneal lymph nodes, rt. femur, and rt. seventh and eighth ribs	osteoblastic metastases	alive

four months' duration. He stated that a week after the onset of his illness, he noted pain and swelling of the left thigh and calf (later attributed to phlebitis) and aching of both testicles, without swelling. When first seen at another hospital, physical examination disclosed enlarged posterior cervical lymph nodes and a large abdominal mass, which was nodular and not well delineated from the liver. Intravenous pyelography showed lateral displacement of the right ureter and right kidney, while the chest roentgenogram exhibited a right pleural effusion. Two exploratory abdominal operations failed to reveal the exact nature of the disease despite biopsies. The retroperitoneal mass was found to be closely adherent to the right kidney and was not excised because of its extent. The patient was subsequently transferred to our institution with a tentative diagnosis of "retroperitoneal sarcoma."

Roentgenograms of the spine showed osteolytic lesions involving the body and pedicles of the second lumbar vertebra. External radiation therapy was administered through three ports: anterior abdominal, posterior abdominal and right lateral, employing a 250 kv. apparatus with Thoraeus filter. A tumor dose of 3,000 r was given to the retroperitoneal mass from August 18, 1949 to September 10, 1949. However, soon after the final treatment the patient's condition deteriorated and he expired on October 3, 1949. A postmortem examination disclosed widespread metastases as well as a small nodule in the right testicle. The histology of the testicular lesion was reported as seminoma but the various metastatic lesions, involving the second lumbar vertebra, the retroperitoneal area and elsewhere, were all reported as metastatic embryonal carcinoma.

DISCUSSION

Osteoblastic changes are distinctly rare in skeletal metastases from malignant testicular tumors. Willis[6] suggests that the

osteolytic and osteoblastic nature of skeletal metastases may depend on different enzymes produced by a particular malignant tumor.

Of a special interest is the fact that several of our patients showed roentgenographic evidence of bone metastases *before* exhibiting pulmonary involvement. An explanation for this phenomenon may be found in Batson's view that the vertebral venous plexus is the major pathway by which tumor emboli disseminate to the axial skeleton, bypassing the lungs.

In Case III the clinical course was unusual. Rarely have we seen an individual with massive metastatic disease prior to clinical detection of the primary testicular tumor. Not so uncommon, however, is the finding of embryonal carcinoma in metastatic foci from a so-called primary seminoma. Whether there lurks in every such lesion an embryonal cell tumor component or whether transformation takes place during the metastatic process remains a controversial question.

SUMMARY

Secondary skeletal involvement in patients with malignant testicular tumors has been documented rather infrequently.

Ten such cases were observed in a group of 167 patients with tumors of the testis seen or treated in the Radiation Clinic of the Veterans Administration Hospital, Bronx, New York, in the past eight years.

Of particular interest are 2 patients with multiple osteoblastic as well as osteolytic metastatic lesions.

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OSTEOBLASTIC BONE METASTASIS IN GASTRO-INTESTINAL AND BRONCHIAL CARCINOIDS*

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THE interest engendered by the recent elucidation of the carcinoid syndrome by Biörck, Axen, and Thorson[2] has resulted in a host of publications dealing with carcinoid tumors. The subject is an extremely intriguing one, particularly so because the experimental and clinical findings have dovetailed so beautifully. A clear picture of the etiology, clinical manifestations, laboratory aspects, and course of this entity has been painted, but additional study is now required to explore the various ramifications of the disease.

Quite recently several publications have presented evidence linking the carcinoid type of bronchial adenoma with intestinal carcinoid. The two-fold purpose of the present report is to lend support to this thesis and to call attention to the occurrence of osteoblastic bone metastasis in both the gastrointestinal and bronchial carcinoid.

GASTROINTESTINAL CARCINOID

Abdominal carcinoid is an epithelial neoplasm with a characteristic histologic appearance. The cytoplasm of the tumor cells frequently contains granules showing an affinity for silver salts; hence the name argentaffinoma. Most workers agree that the tumor arises from the scattered argentaffin cells described by Kulchitsky in the crypts of Lieberkühn in the gastrointestinal tract. Over half of these tumors are found in the appendix. Almost all of the remainder arise in the gut distal to the esophagus, mostly in the small intestine but not infrequently in the stomach and colon. They occasionally originate in the gallbladder, pancreas, mesentery, a Meckel's diverticulum, and even in an ovarian teratoma.

When primary in the appendix, the tumor is generally solitary and very rarely metastasizes. Those arising elsewhere in the abdomen are frequently multiple, and metastasis is not uncommon. MacDonald,[9] in a study of autopsy and clinical records from a number of Boston hospitals, found lymph node metastases in 23.3 per cent and distant metastases to the liver or lung in 16.4 per cent of 146 extra-appendiceal carcinoids. There were no well documented metastases among 207 appendiceal carcinoids. Ariel[1] recorded the sites of metastases in 77 cases of small intestine carcinoids collected from the literature. There were metastases in 42 per cent of the cases: 35 per cent in the abdominal lymph nodes, 13 per cent in the liver, 13 per cent in the mesentery or peritoneum, and 1 case each in the testis and dura. Metastases to the lung, spleen, pancreas, adrenal gland, kidney, brain, ovary, and bone have been infrequently recorded.

The behavior of the abdominal carcinoid is quite remarkable. The primary tumor is slow growing, late in postoperative recurrence, and shows little tendency to destroy the affected organ. The metastases are also sluggish and surprisingly inconsequential in many cases. Following the removal of the primary lesion, the metastases may remain stationary or even regress. The presence of metastases is not incompatible with many years of reasonably healthy existence with this disease.

Bone metastasis, of special interest in the present communication, is apparently quite

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infrequent in abdominal carcinoid, according to the literature. Neither MacDonald nor Ariel, in the reports cited above, mention its occurrence. Dick *et al.*[3] reported an atypical case of abdominal carcinoid of unknown primary site with an osteoblastic metastatic lesion of the humerus which showed "sun-ray" spiculation. Biopsy of the bone lesion showed carcinoid. The tumor tissue contained a large amount of serotonin. Pochaczevsky and Sherman[13] reported a case of gastric carcinoid with multiple bone metastases. They described them as osteolytic but mentioned that one of the lesions showed an expanded sunburst pattern. The one metastasis illustrated in their paper was in the left femur which showed a pathologic fracture. On the reproduction there is a suggestion of sclerosis of the bone surrounding the osteolytic lesion. Jaffe[7] demonstrated a histologically proved osteoblastic metastasis with spiculated margins in the upper humerus. Roentgenologically the lesion resembled an osteogenic sarcoma. The patient showed clinical evidence of the carcinoid syndrome. At autopsy, several years later, carcinoid was also found in the bowel and liver. This case represented the only example of metastatic carcinoid in bone he had encountered. Three additional cases of bone metastasis from intestinal carcinoid have been reported but the roentgen appearances were not described or illustrated.[6,11,12]

The carcinoid syndrome is manifested by a variety of symptoms and signs relating to many of the body systems. The skin may show flushing, regional cyanosis, or various types of rash. Abdominal manifestations are frequent and include pain, diarrhea, liver enlargement, palpable tumor masses, and, occasionally, signs of intestinal obstruction. Repeated episodes of bronchial asthma, auscultatory signs of pulmonic stenosis and/or tricuspid insufficiency, and fluctuation in blood pressure represent the cardiopulmonary effects. An increased incidence of chronic peptic ulcer has been noted in abdominal carcinoid. MacDonald[9] found active gastric or duodenal ulcers in 8 of 21 autopsied cases (38 per cent), the general autopsy incidence of peptic ulcer in the same institution being only 5.5 per cent. Other manifestations include central nervous system disturbances and joint pain.

The key substance in the pathogenesis of the carcinoid syndrome is serotonin (5-hydroxytryptamine). The pharmacology of this unique endocrine secretion has been intensively studied and is now well understood.[14] For our present purposes it need only be pointed out that serotonin is encountered in significantly abnormal amounts solely in carcinoid. The substance itself is increased in the tumor and blood while the concentration of its metabolite, 5-hydroxyindole acetic acid, is elevated in the urine. Although most cases showing the carcinoid syndrome have liver metastases, an occasional case without liver involvement has been reported. Functioning tumors associated with increased levels of 5-hydroxyindole acetic acid in the urine but without clinical manifestations have also been reported.

PULMONARY CARCINOID

Bronchial adenomas constitute about 5 per cent of primary pulmonary neoplasms and are generally found in the primary or secondary bronchi. There are two types, the cylindroma (10–15 per cent) and the carcinoid (85–90 per cent). The carcinoid variety, which concerns us here, is histologically identical with the abdominal carcinoid, except that it rarely contains argentaffin granules. Most authors agree that pulmonary carcinoids originate in the bronchial glands. Hamperl[5] considered the cells in these glands to be closely related to the gastrointestinal Kulchitsky cells. Bronchial carcinoid, like its abdominal counterpart, grows slowly, recurs late after resection, and shows only a moderate tendency to invade surrounding structures. Approximately 10 per cent metastasize but, again, the metastases are usually quite sluggish and often inconsequential. In most instances the metastases are confined to the

tracheobronchial lymph nodes. Metastases to the liver and other distant structures do occur but are not frequent.[8]

As with gastrointestinal carcinoid, bone metastases from pulmonary carcinoid are quite rare. MacBurney *et al.*[8] reported 2 such cases, 1 involving the femur and the other of unstated location. Warner and Southern[17] reported a case of vertebral metastasis. Neither of these authors demonstrated or described the roentgen appearance of the metastatic lesions. Moersch and McDonald[10] recorded and illustrated 1 case with rather extensive osteoblastic involvement of the pelvis and spine.

REPORT OF CASES

The following 2 cases of carcinoid manifested osteoblastic bone metastases. In 1 the primary site was probably within the abdomen; in the other it was in a bronchus.

Case 1. J.W., a forty-seven year old white male, had been in good health until 1951, when episodes of facial erythema, diarrhea, nausea, vomiting, and slight cramping abdominal pain began. The attacks lasted two or three days and recurred at irregular intervals, but averaged about a month apart.

In June, 1955, he was admitted to a hospital during an attack. Red discoloration of the face and upper chest was noted. Roentgen examination of the abdomen revealed multiple areas of increased density in the pelvic bones. Abdominal exploration showed metastatic neoplasm in the liver and omentum but careful examination of the small and large bowel failed to reveal a primary site. Histologic examination of the metastatic tumor showed neoplasm consistent with carcinoid. A serotonin assay was not performed on the tumor tissue.

He was admitted to the Cincinnati Veterans Hospital in May, 1957, because of recurrence of the symptoms. Physical examination revealed diffuse erythema of the face and trunk. The lips and the tip of the nose showed a bluish discoloration. Examination of the chest was normal. There were no cardiac murmurs. The liver was palpable four finger-breadths below the right costal margin. A fascinating clinical feature was the patient's ability to reproduce or aggravate his symptoms and signs by bending at the waist. Massaging the abdomen did not produce the same effect.

Fig. 1. Case 1. Extensive coalescent and discrete osteoblastic nodules in the pelvis and lumbar spine.

The white blood cell count was 26,000 per cu. mm., with 89 per cent neutrophils. Subsequently these returned to normal levels. Other laboratory studies were not remarkable except for a persistent marked increase in the urinary level of 5-hydroxyindole acetic acid.

Roentgenograms of the bony skeleton showed numerous discrete and coalescent osteoblastic nodules in the pelvis, spine, and ribs (Fig. 1 and 2). The heart and lungs were normal. Barium enema examination, upper gastrointestinal series, and small bowel study failed to reveal evidence of a primary neoplasm. Gallstones were present. Biopsy of the ilium showed metastatic neoplasm thought to represent carcinoid, with osteoblastic reaction in the bone. Argentaffin granules were not present in the tumor cells. A needle biopsy of the prostate showed no evidence of neoplasm.

The patient was discharged from the hospital without further treatment. When last seen in June, 1959, he was working regularly as a watchman. He complained of weakness and occasional episodes of flushing and diarrhea. Roentgenograms revealed minimal progression of the osteoblastic lesions.

Case 11. B.W., a sixty-one year old white female, was first seen in 1951 with symptoms of

Fig. 2. Case I. Lateral roentgenogram showing osteoblastic metastases in the dorsal vertebral bodies.

cough, fever, sweating, and anemia. Family history revealed that her mother had died of cancer of the lung. A chest roentgenogram demonstrated a nodular density about 4 cm. in diameter in the left lower lobe near the hilus. Bronchoscopy showed a smooth vascular tumor 4 mm. in diameter in the left lower lobe bronchus. In December, 1951, resection of the left lower lobe was performed. The bulk of the tumor extended, iceberg-like, into the adjacent lung. There was no evidence of lymph node metastasis or of extension beyond the lower lobe but tumor tissue was found in the bronchus within 3 mm. of the line of resection. Histologic examination revealed a bronchial adenoma of the carcinoid type.

Fig. 3. Case II. Extensive coalescent osteoblastic metastases in the right ilium. The left ilium and lumbar spine show less severe involvement. A metastatic nodule is visible in the intertrochanteric region of the left femur.

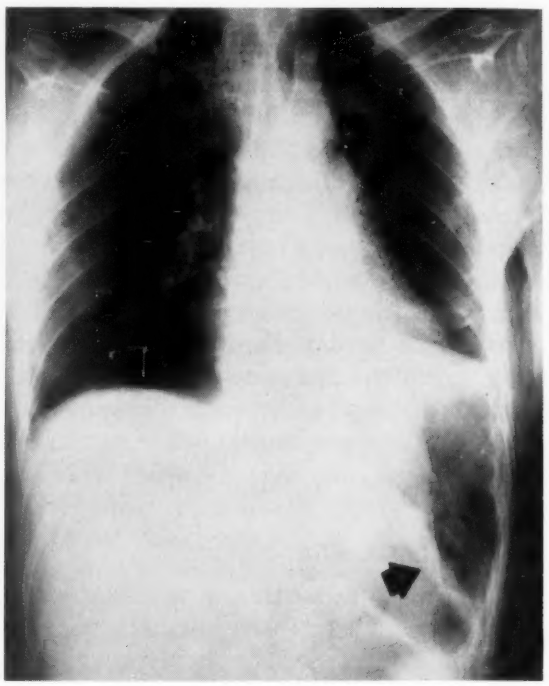

Fig. 4. Case II. Diffuse osteoblastic involvement of the left eleventh rib (arrow). The chest is otherwise normal except for the sequelae of the left lower lobectomy.

The patient remained well, except for persistent anemia, until January, 1954, when right hip pain developed. A roentgenogram showed an ill-defined area of increased density in the right ilium adjacent to the sarcroiliac joint. In April, 1954, she noted progressive impairment of vision in the right eye. Examination revealed a retinal detachment. At about the same time, left lower chest pain appeared and roentgenograms now showed irregular sclerosis of the eleventh rib on the left. Shortly thereafter the right eye was enucleated in another city and a histologic diagnosis of malignant melanoma of the uveal tract was made.

During the next two years she complained of a chronic cough. No evidence of recurrence of the lung tumor was demonstrated roentgenographically or bronchoscopically. In June, 1956, she was admitted to the hospital because of cramping abdominal pain, backache, cachexia,

and subcutaneous scalp nodules. Roentgenograms showed extensive osteoblastic metastatic deposits in the pelvis, spine, and eleventh rib (Fig. 3, 4 and 5). Biopsy of one of the subcutaneous nodules revealed metastatic neoplasm suggestive of malignant melanoma. Her course was slowly downhill and she died in February, 1957.

At autopsy, recurrence of the bronchial adenoma was found in the stump of the left lower lobe bronchus. There were metastatic nodules in the hilar lymph nodes, liver, kidney, dura, and subcutaneous tissue. The ilium, sacrum, vertebrae, and eleventh left rib showed marked areas of osteosclerosis. The gastrointestinal tract was entirely normal. Histologically, all of the nodules, including those in the bone, showed the same type of neoplasm. The appearance was identical with that of the eye tumor, the scalp nodule, and the original bronchial adenoma, and all showed the typical appearance of carcinoid. The final diagnosis was bronchial adenoma of the carcinoid type with widespread metastases. Serotonin studies were not performed on either the necropsy or biopsy material, nor was the urine studied during life for 5-hydroxyindole acetic acid.

Fig. 5. Case 11. Lateral roentgenogram of cervical spine showing osteoblastic changes in the body, neural arch, and spinous process of the axis.

DISCUSSION

Recent evidence indicates a close relationship between abdominal and bronchial carcinoid. At least 6 cases of bronchial adenoma of the carcinoid type have been reported in which some of the manifestations of the carcinoid syndrome were present.[4,14,16,17] In 4 of the cases, the pulmonary lesion had been resected prior to the recognition of the syndrome, and hepatic metastases and increase in the urinary 5-hydroxyindole acetic acid were subsequently demonstrated. In each of the other 2 cases, although urinary studies were not performed, serotonin was found in the tumor itself. Autopsy was performed in 2 of the 6 cases and in neither was a primary neoplasm found in the gastrointestinal tract. Metastatic liver involvement was demonstrated in 1 while an apparently unrelated hepatoma was found in the other. In 3 cases barium studies and/or abdominal exploration failed to reveal evidence of gastrointestinal carcinoid.

The possibility that the bronchial tumor in these cases might represent metastasis from intestinal carcinoid of insignificant size must be given consideration. The rarity of lung metastases from abdominal carcinoid and the infrequency, in general, of metastasis to the wall of a bronchus make this extremely unlikely.

As stated earlier, bone metastasis in abdominal and bronchial carcinoid is infrequent. It is quite impressive, then, to note that in 3 of the 4 recorded cases of gastrointestinal carcinoid and in both of the reported cases of bronchial carcinoid in which the character of the bone lesions was described (including our own cases) the metastatic lesions were osteoblastic in nature. Even in the fourth case of abdominal carcinoid, the metastases may have had a sclerotic component. This intriguing association of osteoblastic metastasis with carcinoid has not been recognized previously.

The similarities between abdominal and

pulmonary carcinoid are so striking that they merit increased attention. Apart from the presence of argentaffin granules, they are identical histologically. Even with respect to the granules the difference is not absolute, since they are occasionally found in bronchial and are frequently absent in abdominal carcinoids. In Case 1 (abdominal carcinoid) the bone metastases, at least, did not show them.

The slow rate of growth of the intestinal form is paralleled by that of pulmonary carcinoid. The incidence of metastasis to the regional lymph nodes and to more distant structures is about the same in both conditions and the frequently prolonged survival of individuals suffering from metastases from either of these primary sites is a feature seldom encountered with other metastatic tumors. The cases of bronchial carcinoid, cited earlier, with the clinical manifestations of the carcinoid syndrome and increased amounts of serotonin or its derivatives in the tumor or urine afford strong evidence that abdominal and bronchial carcinoid are closely allied.

The fact that metastases from certain tumors, such as lymphoma and carcinoma of the breast and prostate, are frequently osteoblastic, whereas those from other tumors are rarely so, makes the relatively high percentage of osteoblastic metastases in carcinoid even more significant. Since both abdominal and pulmonary carcinoids seem to show this propensity, another link can be added to the chain of evidence which indicates that they are closely related entities.

SUMMARY

Since the elucidation of the carcinoid syndrome, evidence has been accumulated indicating a close relationship between abdominal carcinoid and bronchial adenoma of the carcinoid type. The present report deals with 2 cases of osteoblastic bone metastases, one arising from an abdominal carcinoid and the other from a bronchial carcinoid. Although bone metastasis in either condition is quite rare, a review of case reports in the literature indicates that a high percentage of those described are osteoblastic in nature. This offers supportive evidence for the thesis that abdominal and bronchial carcinoid are related entities.

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PRIMARY HYPERPARATHYROIDISM*

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SEVERE generalized disease of the skeletal system may be caused by primary or secondary hyperparathyroidism. The primary disease may be due to (1) adenoma, (2) diffuse hypertrophy and hyperplasia, or (3) rarely, carcinoma of the parathyroid glands. Secondary hyperparathyroidism may be associated with certain types of advanced renal disease and with severe osteomalacia.

Bone changes need not be manifest in primary hyperparathyroidism. Thus in the two largest series of reported cases,[3,37] bone disease was not demonstrable in 10 and 30 per cent of patients, respectively. This is shown in Table I.

Primary hyperparathyroidism is a potentially fatal disease which is curable by surgical resection of the adenoma or the hyperplastic gland. Early recognition of bone changes is of great importance in establishing the diagnosis at a time when correct management may minimize morbidity and incapacitation.[7,12,13] It is obvious that radiologists, as well as the internists, orthopedists, urologists and dentists have the opportunity and the responsibility of making the diagnosis at an early stage. The secret is to keep the possibility in mind until the diagnosis is confirmed or excluded by other studies.

Seven cases of primary hyperparathyroidism comprise this series, all proved to have parathyroid adenoma at operation. Four patients had extensive bone disease; one had a renal stone and early bone changes; another had recurrent renal stones and suspected bony abnormalities; and one had no demonstrable bone or kidney disease. Included is another patient whose roentgenograms demonstrated renal calcinosis and healed cystic lesions in the ilium and pubis ten years after parathyroidectomy for hyperparathyroidism. Information about these patients is summarized in Table II.

The age distribution found in larger series[11,25] shows the highest incidence in the third, fourth and fifth decades. In our series, there are 3 patients in the fifth and 1 patient each in the second, third, fourth, sixth and seventh decade.

The female to male ratio has been found to be about 3:1 in most large series.[11,25] We have 7 females to 1 male.

In each of our patients a single benign adenoma was found, with two located in the upper mediastinum, which is a common site.[17] There is no case of diffuse hyperplasia or multiple adenoma in our series. Castleman and Mallory[11] analyzed 160 cases. They found adenomas in 86.4 per cent and hyperplasia and hypertrophy in 13.6 per cent. Nine per cent of the adenomas involved more than one gland.

TABLE I

INCIDENCE OF BONE AND RENAL DISEASE IN PRIMARY HYPERPARATHYROIDISM

	Author and Institution	
	Albright and Reifenstein[3] Massachusetts General Hospital	Pugh[37] Mayo Clinic
No. of Patients with Bone Disease	11	29
No. of Patients with Renal Disease	28	33
No. of Patients with Both	24	9
No. of Patients with Neither	1	5
Total	64	76

* From the Department of Radiology, Baylor University College of Medicine, and Jefferson Davis Hospital, Houston, Texas.

TABLE II

CASE SUMMARIES

Case No.	Patient	Age	Sex	Race	Parathyroid Gland		Renal Disease	Bone Disease	Remarks
					Pathology	Location			
1	D.J.	23	F	N	Single benign adenoma	Left lower pole of thyroid	None demonstrable	Advanced	Expanding cyst-like lesion in 1 rib and resorption of ends of clavicles in routine chest roentgenogram 3 years ago
2	N.M.	49	F	N	Single benign adenoma	Left lower pole of thyroid	Renal stone	Advanced	Sustained multiple fractures in minor automobile accident 5 years ago. Bedridden since
3	O.B.	47	F	LA	Single benign adenoma	Right superior anterior mediastinum	Renal stone	Advanced	Kidney stones for years. Very little symptoms otherwise. Cares for 8 people in her house
4	B.H.	57	F	W	Single benign adenoma	Left lobe of thyroid	Renal calcinosis	Advanced	Low back pain. Recent bilateral femoral fracture
5	B.S.	38	F	N	Single benign adenoma	Right superior anterior mediastinum	Renal stone	Relatively early	"Giant cell epulis" of right maxilla led to correct diagnosis
6	J.P.	41	F	W	Single benign adenoma	?	Renal calcinosis	Healed	Parathyroid adenoma removed 10 years ago
7	R.C.	15	M	LA	Single benign adenoma	Left lower pole of thyroid	Renal stone	Very early changes	Drinks 2 quarts of milk a day
8	M.D.	68	F	N	Single benign adenoma	Right lobe of large nodular goiter	None demonstrable	Changes not differentiable from osteoporosis	Parathyroid adenoma found in goiter specimen incidentally

Carcinoma is a rare cause of hyperparathyroidism.[11,42] Albright and Reifenstein[3] stated that they knew of only 3 cases.

The incidence of both renal and skeletal disease is higher in our patients than in the Massachusetts General Hospital[3] or Mayo Clinic[37] series (Table 1). This is probably because our patients were in a more advanced stage of the disease when first diagnosed.

THE CLINICAL SYNDROME

The symptomatology of primary hyperparathyroidism has been well documented[3,4,5,8,25,28,29] and may be divided into three groups: (1) that of hypercalcemia *per se;* (2) that of kidney involvement; and (3) that of bone disease.

The onset of the disease is insidious. The course is protracted. The symptoms develop and progress slowly and steadily. It may be many months or years before the patient seeks medical care. Even then, the diagnosis may not be made immediately, unless the physician entertains a high degree of suspicion and proceeds with appropriate laboratory studies.

1. *Symptoms of Hypercalcemia.* Although hypercalcemia is one of the cardinal chemical changes in primary hyperparathyroidism, symptoms due to hypercalcemia *per se* are frequently not recognized as such. Because they tend to be vague and nonspecific, they are often attributed to other ailments. These symptoms are neuromuscular and gastrointestinal in origin, due primarily to increased calcium ion concentration in the extracellular fluids. Muscular hypotonia and weakness are prominent symptoms and may be very marked. Muscle and joint aching pains may simulate rheumatism. Nausea and vomiting may be troublesome or intractable. Constipation may be stubborn. Anorexia is common. The patient gradually and progressively becomes bedridden and wasted.

2. *Symptoms of Renal Involvement.* The

renal symptoms are secondary to increased urinary excretion of calcium and phosphorus. This leads to polyuria accompanied by thirst and polydipsia. The occurrence of nephrolithiasis and nephrocalcinosis is attended by renal colic, hematuria, symptoms of obstruction and infection, and eventually progressive renal insufficiency.

3. *Symptoms of Bone Involvement.* The symptoms of bone disease in hyperparathyroidism vary with the severity of the involvement. When the changes are sufficiently advanced, there may be deep pains and bone tenderness. In more advanced stages, pathologic fractures and various types of deformities are frequent findings. The patient is racked with intractable pain, crippled and bedridden.

THE PATHOPHYSIOLOGY AND CHEMICAL CHANGES

Our knowledge of the physiology of bone and calcium and phosphorus metabolism and of the locus and mechanism of parathyroid hormone action has accumulated since the time of von Recklinghausen. A number of excellent monographs and reviews bearing on these topics have appeared in the literature.[3,6,14,15,23,24,26,27,30,31,32,36]

For our purpose, the following considerations may suffice:

1. A number of important physiologic functions are dependent upon calcium and phosphate ions in the body fluids (Table III and IV). Normally, the concentrations of these ions in the serum are rigorously maintained within rather narrow limits.

2. The skeletal system, composed of organic matrix and inorganic bone salts, contains approximately 99 per cent and 66 per cent of the total body calcium and phosphorus, respectively. As a storehouse of these minerals, the bone serves as a ready source of calcium and phosphorus for the body fluids.

Table III

PHYSIOLOGIC FUNCTIONS OF CALCIUM IONS

1. Neuromuscular activity
2. Myocardial contraction
3. Blood coagulation
4. Capillary permeability
5. Bone formation (storage and reserve)

Table IV

PHYSIOLOGIC FUNCTIONS OF PHOSPHATE IONS

1. Acid-base balance
2. Energy metabolism
3. Intermediary metabolism
4. Enzymes and co-enzymes
5. Cellular structures
6. Bone formation (storage and reserve)

3. The internal secretion of the parathyroid glands, parathormone, is intimately concerned with the regulation of calcium ion concentration of the serum. By mobilizing bone salt from the skeletal system, it regulates the supply of calcium to the extracellular fluids. The functional activity of the parathyroids is in turn regulated by the serum calcium level by a feed-back mechanism. A falling level stimulates the parathyroids to increased production of parathormone.

4. There has been considerable controversy with regard to the primary site of action of parathormone. One school[1,2,3] believes the primary locus of parathormone action is on the renal tubules, causing interference with reabsorption of phosphate ions and secondary resorption of bone. Another school[14,15,31,32] advocates that the bone resorption is the primary action. More recently, a compromise view[24,26,33] has been advanced that the parathyroid hormone may be comprised of two fractions, one acting on the renal tubules and the other on the bone, directly and primarily.

5. Although the exact mechanism of action of parathormone on the bone remains to be clarified, the result is osteoclastic resorption of bone with mobilization of bone salt to the circulation.

It should be emphasized that, under normal circumstances, parathyroid function is finely regulated by serum calcium

concentration. In the so-called isoparathyroid state, the action of parathormone is subtle and not apparent.

6. In the pathologic state of hyperparathyroidism, however, the feed-back mechanism breaks down; the abnormal and unregulated increase in the production of parathormone causes uncontrolled and exaggerated actions on the target organs resulting in characteristic chemical changes. These are (a) hyperphosphaturia; (b) hypophosphatemia; (c) hypercalcemia; and (d) hypercalcuria.

Increased urinary excretion of phosphate is caused by inhibition of tubular reabsorption. The demonstration of diminished tubular reabsorption of phosphate (TRP) has been found useful in the early recognition of hyperparathyroidism.[22] The loss of phosphate is of such magnitude that the serum inorganic phosphorus level is diminished in spite of mobilization of large amounts of phosphate and calcium from the skeletal system. The extracellular fluids are flooded with calcium from osseous resorption and urinary excretion of calcium may be greatly increased. This combination of chemical findings is not seen in any disease other than primary hyperparathyroidism.

7. In the presence of bone disease, the serum alkaline phosphatase is increased, indicating increased osteoblastic activity, a compensatory response to increased stress caused by osteoclastic resorption.

8. With progressive renal damage due to renal calculi or calcinosis and their complications, azotemia, acidosis, and eventually retention of phosphate may occur. The latter may mask hypophosphatemia and reduce the hypercalcemia.

The chemical findings in our patients are presented in Table v.

THE ROENTGEN CHANGES

It is well known that alterations of structural integrity and/or calcium content of bone must be sufficiently great before the changes become demonstrable in the roentgenograms. During the protracted course of primary hyperparathyroidism, a generalized progressive bone disease develops. It is to be expected that in the very early stage bone changes would not be demonstrable in roentgenograms. As the disease progresses, however, more roentgenographic changes become recognizable with greater ease and confidence.

Pathologically,[28,30] the basic lesion of osteitis fibrosa cystica is increased osteoclastic resorption of bone and fibrosis of marrow spaces. This process is generalized; every bone is affected, although the degree of involvement may vary. With progressive de-ossification, cysts and cyst-like lesions develop. As the bones become weakened and softened, pathologic fractures and deformities occur. In response to increased stress, osteoblastic reparative processes are stimulated. Unlike osteomalacia, the osteoid tissue formed is calcified and ossified. By and large, however, the laminal bone,

Table V

SUMMARY OF CHEMICAL FINDINGS

Case No.	Before Operation			Immediately after Operation	Long Term Follow-Up
	Serum Ca	Serum P	Serum Alk. Phos.		
1	12.8	1.6	42.0	Moderate hypocalcemia and latent tetany readily controlled by calcium therapy in all cases	Elevated serum alkaline phosphatase persisted for several weeks or months; serum Ca and P levels returned to normal soon after operation
2	12.1	2.4	41.3		
3	12.2	—	67.0		
4	16.7	2.8	14.4		
5	—	—	24.3		
6	11.2	3.0	—		
7	15.8	2.5	4.8		
8	—	—	—		

which is resorbed, is replaced with woven bone, which is hastily formed in the immediate vicinity of destruction. The architecture of the bone is distorted and its strength is only partially restored.

The roentgen appearance of advanced osteitis fibrosa cystica has been well documented.[9,10,16,19,20,21,25,37,38,40] The *generalized* decalcification and the presence of *cyst-like* lesions are stressed as predominant findings. The recognition of early changes, however, has not received sufficient emphasis.

With our material, we propose: I, to recapitulate the advanced changes that are pathognomonic; II, to present the early changes that are characteristic and diagnostic; III, to demonstrate the very early changes that should arouse suspicion; and IV, to present changes associated with healing. A summary of the bone changes seen in our patients is shown in Table VI.

I. ADVANCED BONE CHANGES THAT ARE PATHOGNOMONIC

A. *Subperiosteal and Endosteal Resorption of Compact Bone*

1. These changes are best seen in the tubular bones of the hand, especially in the middle phalanges of the fingers. The subperiosteal surface of the cortex becomes fuzzy, ragged and irregularly finely spiculated in appearance. The sharp definition and smooth contour are lost. Endosteal resorption is evidenced by a scalloped contour of the inner margin of the cortex. The thickness of the cortex is markedly diminished as a result of resorption from both surfaces. Increased resorption of the tuft of distal phalanges presents an irregular, moth-eaten appearance and eventually complete dissolution of the tuft or even the entire terminal phalanx (Fig. 1*A*).

2. The cortex of the long bones of extremities may show similar findings. The favorite sites are the distal ulna and upper tibia. As a rule, the subperiosteal resorptive changes are not as spectacular as in the phalanges. The scalloped endosteal margin and the marked thinning, however, are no less apparent (Fig. 2).

3. A similar process affects all other bones. The cortical bone of the inferior aspect of the distal third of the clavicle, the vertebral bodies, the ribs, the skull, etc., are involved in most cases of advanced disease.

B. *Subperiodontal Resorption of Lamina Dura*

The loss of lamina dura (Fig. 3 and 6) may be considered as a special form of subperiosteal resorption. The lamina dura is the cortex of the dental socket and the

TABLE VI

SUMMARY OF ROENTGENOGRAPHIC FINDINGS

Case No.	Generalized Decalcification	Subperiosteal Resorption of Hand	Resorption of Clavicle	Resorption of Lamina Dura	Resorption of Symphysis Pubis	Diploic Resorption of Skull	Expanding Osteoclastoma	Cyst-like Lesions Elsewhere	Pathologic Fractures	Deformities
1	Very marked	Typical	Ends and distal shaft	Complete	Marked	Typical	One rib	Multiple	Multiple repeated	Hips
2	Very marked	Typical	Ends and distal shaft	Complete	Moderate	Typical	—	Multiple	Multiple repeated	Thoracic cage, spine, pelvis, hips
3	Marked	Typical	Distal shaft	No teeth	—	Typical	Several ribs and pelvis	Multiple	Pubis (healed)	Spine
4	Very marked	Typical	Distal shaft	No teeth	—	Typical	One metacarpal	Multiple	Multiple repeated	Spine, thoracic cage, hips
5	Moderate	Slight	Distal shaft	Incomplete	—	Slight	Maxilla (resected)	Mandible	—	—
6	None (10 years postoperatively)	—	—	—	—	—	—	Pubis and ilium (healed)	—	—
7	Minimal	—	Distal shaft	—	?	—	—	—	—	—
8	Osteoporosis	—	—	Bad teeth, many missing	—	Osteoporosis	—	—	—	—

Fig. 1. Case 2. (*A*) Characteristic subperiosteal and endosteal resorption and multiple cyst-like lesions of all tubular bones and resorption of tufts of terminal phalanges. (*B*) Recalcification one year after surgical removal of parathyroid adenoma.

periodontal membrane is a special form of periosteum continuous with that of the alveolar process. Though the loss of lamina dura has been observed in other diseases,[3,38] it nevertheless is a valuable sign and frequently calls attention to the possibility of hyperparathyroidism.[3]

C. *Subchondral Resorption of Clavicle and Symphysis Pubis*

The resorption of the ends of clavicles has been reported[34] as a characteristic sign of hyperparathyroidism. This occurred in 2 of our cases (Fig. 4). Similar resorption of subchondral cortical bone of the symphysis pubis was noted in 2 of our patients (Fig. 5 and 10). The latter finding has not been described previously.

The constant stress due to movements of the shoulder joints has been suggested as contributory to the subchondral resorption of the ends of the clavicles.[34] The symphysis pubis is subject to no less stress. A similar process may contribute to the resorption of articular cortex of the vertebral bodies, the phalanges and other bones.

D. *Trabecular Resorption of Spongy Bone*

1. Osteoclastic resorption of the diploic structures of the cranial bone has received much attention. Roentgenographically, the skull presents a diffuse loss of density with a characteristic miliary, stippled, granular, wooly or mottled appearance. As a rule, there are cyst-like areas of varying sizes. The outer and inner tables are markedly thinned and particularly the outer table may not be demonstrated with certainty.

Fig. 2. Case 1. Marked resorption of cortical bone and spongiosa, multiple cyst-like lesions, and scalloped inner margin of cortices especially noticeable in distal end of tibia.

Fig. 4. Case 1. Subchondral resorption of the end of the clavicle and subperiosteal resorption of the inferior aspect of the distal clavicle and of the ribs.

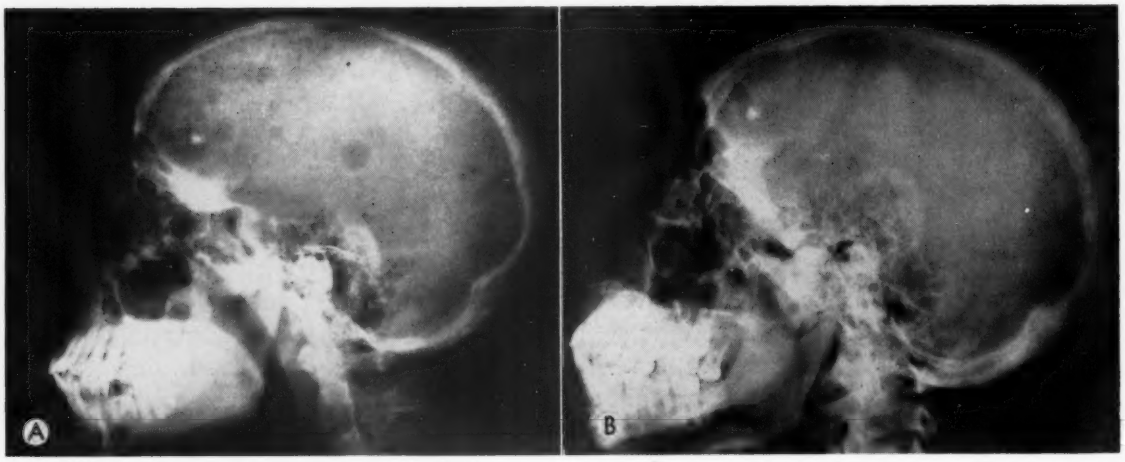

Fig. 3. Case 1. (*A*) Characteristic granular type of resorption, marked thinning of outer table, apparent thickening of diploë and multiple cyst-like lesions in calvarium. Note also loss of lamina dura. (*B*) Recalcification one year after surgical removal of parathyroid adenoma with pagetoid-like rounded areas of increased density replacing the previous cyst-like radiolucencies.

Fig. 5. Case 1. Subchondral resorption of articular cortices of symphysis pubis, multiple cyst-like lesions, and bilateral pathologic fractures of femoral necks.

The diploë usually appears to increase in thickness (Fig. 3, 6 and 13).

2. Trabecular resorption involves all spongy bones. Besides the skull, the roentgenographic changes are seen best in the small bones of the hands (Fig. 1), the

Fig. 6. Case 2. Stippled appearance of the skull, decrease in density of the outer table, loss of superior margins of the orbits, and loss of the lamina dura.

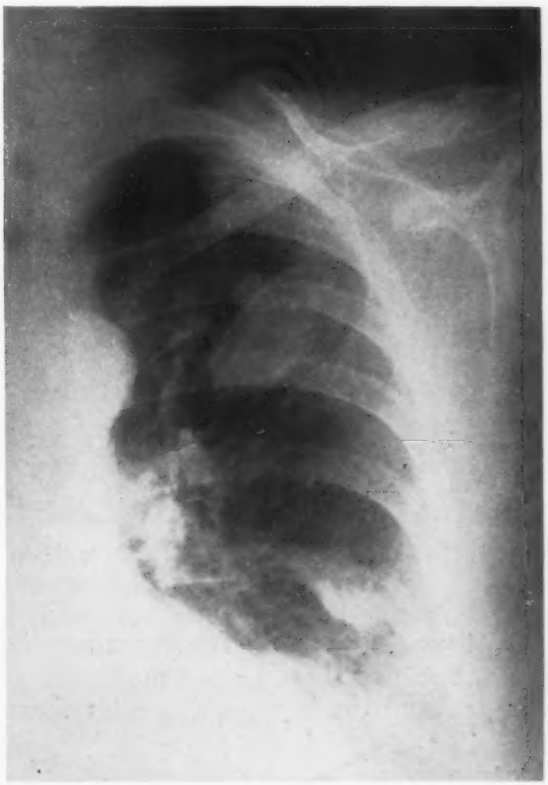

Fig. 7. Case 3. Expanding lesion of anterior segment of the left second rib, healed fractures of the clavicle and rib and marked resorption of all bones. Several similar expanding lesions are present in the ribs on the right side.

ribs (Fig. 4, 7 and 12), the vertebral bodies (Fig. 8, 9, 14, 19 and 20), the pelvis (Fig. 5 and 10), the mandible (Fig. 13) and the ends of the long bones (Fig. 2). As an increasing number of trabeculae is thinned and completely destroyed, the trabecular spaces are widened and may be filled with fibrous tissue or fluid. The remaining trabeculae appear coarsened and lacy as they stand out in contrast to the fibrotic spaces. With further progression of the resorptive process, the trabecular structural pattern becomes increasingly indistinct and may present a miliary, mottled, granular or even a ground glass appearance.

3. Cysts, brown tumors and osteoclastomas are the end results of extensive localized resorption of bone. Roentgenographically, they all appear as cyst-like lesions. They may be seen in any bone. The

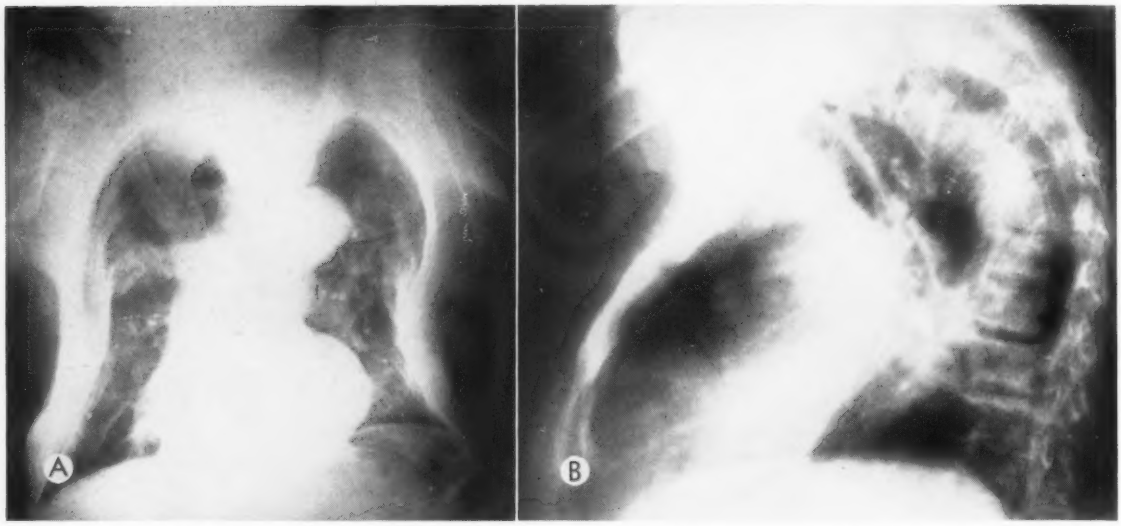

Fig. 8. Case 2. (*A*) Severe deformity of the thoracic cage secondary to marked decalcification of the ribs. (*B*) Marked kyphosis due to softening of the vertebrae.

jaw, the pelvis and the femur are favorite sites. Some of these lesions may grow in size, expand the bone, and present themselves as palpable tumors. This occurs especially in the maxilla, mandible (Fig. 13) and the ribs (Fig. 7). These lesions may be mistaken for giant cell tumors or fibrous dysplasia clinically and pathologically.[18,35,43] One patient in our series (Case 5) had partial resection of the maxilla for "giant cell tumor" before the diagnosis of hyperparathyroidism was suspected and established at surgery. Two other patients had expanding cyst-like lesions in the ribs, and one patient had a similar lesion in a metacarpal bone.

E. *Secondary Bone Changes*

The secondary changes of advanced osteitis fibrosa cystica are (1) marked softening of the bones; (2) pathologic fractures; and (3) various deformities. The softening of the skull may lead to basilar invagination. The softening of the vertebral bodies results in wedging, flattening, kyphosis and scoliosis with loss of stature. The unyielding nucleus pulposus causes typical biconcave deformity of adjacent vertebrae (Fig. 8 and 9). The pelvis may

Fig. 9. Case 2. Marked decalcification, cyst-like lesions and biconcave deformities of lumbar vertebrae. Solitary renal calculus.

be markedly distorted (Fig. 10). The thoracic cage may cave in (Fig. 8). Pathologic fractures may occur in any bone with minimal or no trauma. The femoral neck and the ribs are common sites. (Fig. 5, 10 and 11).

II. LESS ADVANCED BONE CHANGES THAT ARE DIAGNOSTIC

The bone changes described above are those of advanced bone disease associated with primary hyperparathyroidism which must have been present for a number of years. The changes are pathognomonic and should offer no difficulty in diagnosis.

During the insidious development of the bone lesions, it is conceivable that less advanced changes may be more difficult to recognize at an earlier stage. Such is the case in one of our patients whose case history is briefly presented.

FIG. 10. Case 2. Extensive cyst-like lesions and marked deformity of pelvic bones, protrusio acetabuli, and subchondral resorption of symphysis pubis.

CASE 5. B.S., a thirty-eight year old housewife, first noticed a lump in the right side of the upper gum extending to the roof of the mouth.

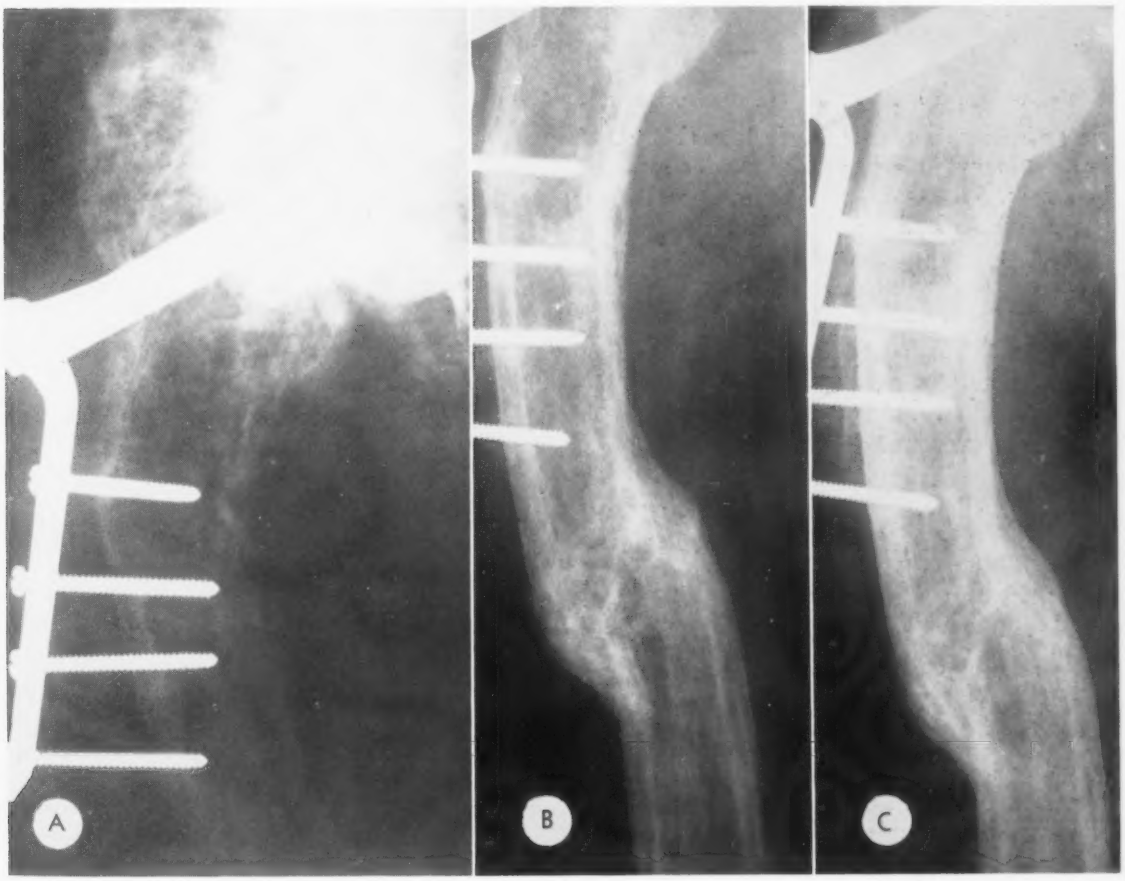

FIG. 11. Case 4. Extreme resorptive changes, numerous cyst-like lesions and pathologic fracture of femur before operation (*A*), and progressive healing and recalcification after surgery (*B* and *C*).

Fig. 12. Case 5. Subperiosteal resorption of distal clavicle, coarsening of trabecular pattern and thinning of cortices of ribs.

She experienced some toothache. The right upper molar was extracted, but the wound healed slowly. The gum swelled with intermittent pain. A giant cell epulis of the maxilla was diagnosed clinically. A partial resection of the right maxilla was performed. The histologic examination was reported as "giant cell epulis of bone" with possibility of hyperparathyroidism.

Fig. 13. Case 5. Partial loss of lamina dura, large cyst-like lesion in mandible, and osteoporotic appearance of calvarium, although the characteristic stippling is lacking.

She was admitted to M. D. Anderson Hospital and was found to have bilateral renal calculi, and a pressure defect to the right of the esophagus at the level of T-1. A parathyroid adenoma was removed at surgery. The roentgenographic findings were as follows: (1) generalized moderate decrease of bone density; (2) thinning of cortices of bones of hands and feet without the characteristic appearance of advanced subperiosteal resorption; (3) minimal but recognizable resorption of the inferior aspects of of the distal clavicle; (4) minimal but recognizable evidence of resorption of the medial aspect of proximal tibia; (5) partial loss of the lamina dura; (6) cystic lesion in the maxilla and a similar but less conspicuous lesion in the mandible; (7) slight to moderate coarsening of trabecular structures in the skull, mandible, spine, ribs and long bones; (8) bilateral renal

Fig. 14. Case 5. Renal calculi and coarsening of trabecular pattern of lumbar vertebrae and pelvic bones.

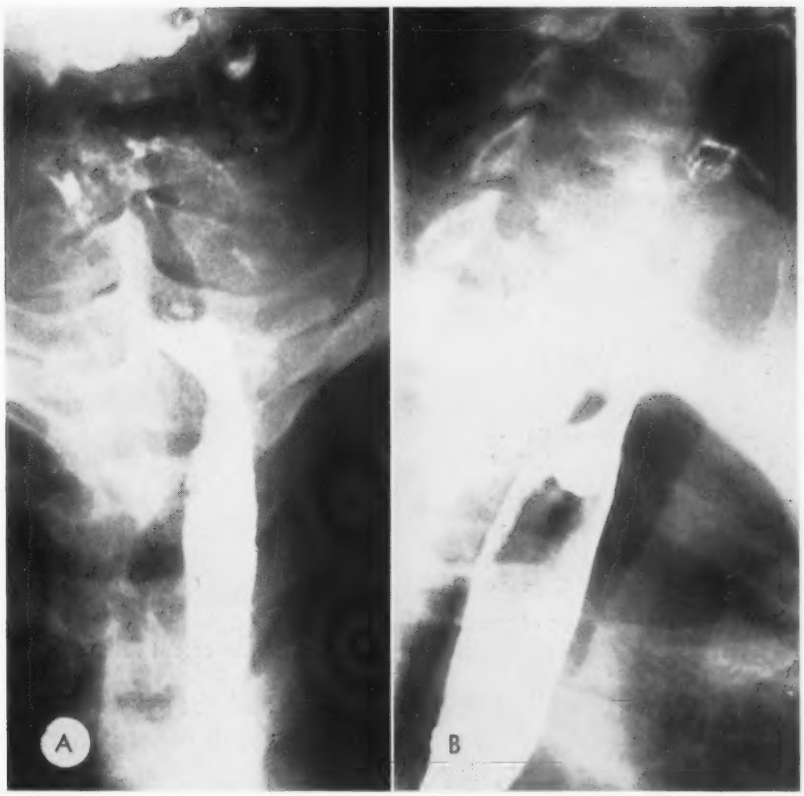

Fig. 15. Case 5. (*A* and *B*) Impression on barium-filled esophagus by parathyroid adenoma, which was resected and measured 3×2 cm.

calculi; and (9) pressure on the esophagus. Some of these changes are shown in Figures 12–15.

Although these less advanced lesions are not in themselves pathognomonic, the suspicion aroused by recognition of such changes should lead to a correct diagnosis.

III. VERY EARLY BONE CHANGES THAT SHOULD AROUSE SUSPICION

In another patient in our series, Case 7, the diagnosis of hyperparathyroidism was made on the basis of recurrent renal stones and typical blood chemical changes. Bone disease was not suspected in the preoperative roentgenograms. However, in reviewing these roentgenograms and comparing them with the follow-up roentgenograms taken two years after surgery, certain changes are demonstrated which we believe represent very early lesions of osteitis fibrosa (Fig. 16 and 17). These changes are: (1) minimal coarsening of trabecular pattern in the vertebrae and in the pelvic bones; (2) minimal resorption of the distal clavicle; (3) possible resorption of the symphysis pubis; (4) thinning of the cortical plate of lumbar vertebral bodies; and (5) definite improvement of the above changes two years after surgical removal of a parathyroid adenoma.

The preoperative findings in themselves are indeed very minimal. With the diagnosis established, they seem more suggestive. With the postoperative improved appearance for comparison, they seem even more meaningful.

IV. THE HEALING OF BONE LESIONS AFTER SURGERY

In hyperparathyroidism bone disease develops because of greatly increased osteoclastic resorption exceeding osteoblastic repair. When the cause is removed by surgery, the pathologic resorptive de-

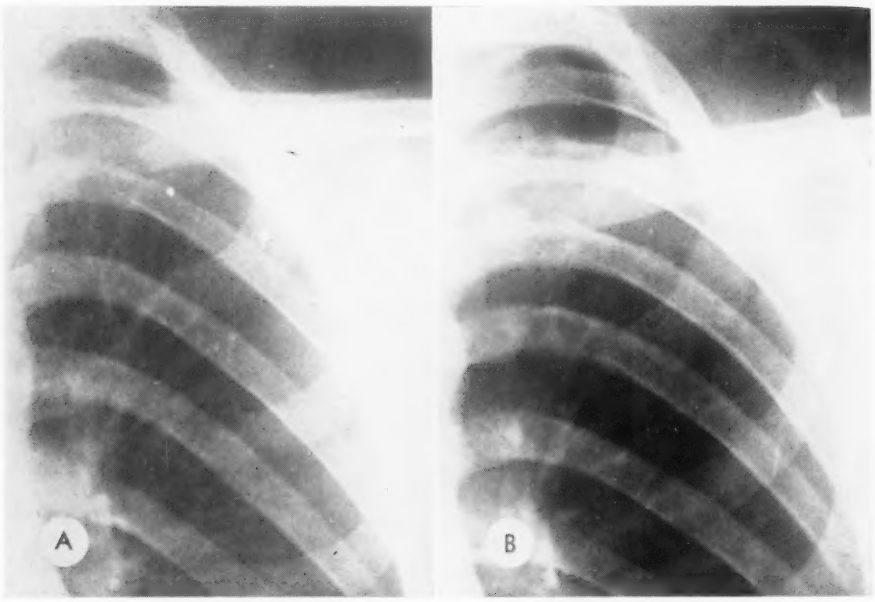

Fig. 16. Case 7. Comparison of preoperative (*A*, 1955) and postoperative (*B*, 1957) appearance. Minimal subperiosteal resorption and improvement after surgery.

struction of bone is stopped while osteoblastic response continues as long as the stress stimulus remains operative. The rapidity of reconstruction of bone is dependent upon the supply of building material, especially calcium, the extent of destruction and the degree of depletion of the skeletal system of its mineral content. The healing of bone lesions may become evident a few months after operation. Complete healing, however, may require many years. Some of these changes are illustrated in Figures 1, 3 and 11.

Cystic lesions, especially the larger ones, may fail to heal completely after all other

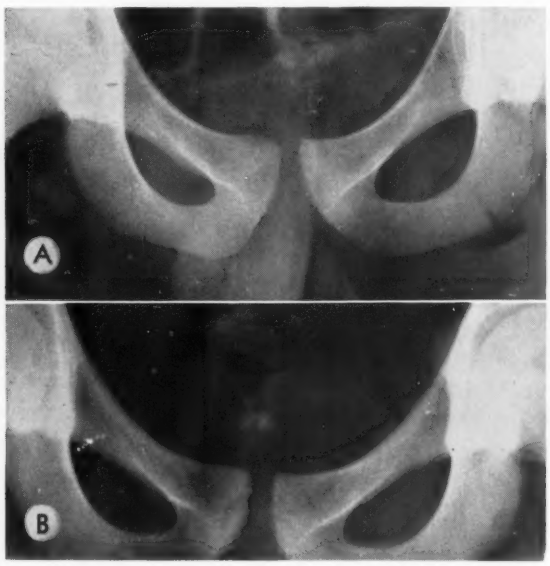

Fig. 17. Case 7. Comparison of preoperative (*A*, 1955) and postoperative (*B*, 1957) appearance of symphysis pubis. Minimal subchondral resorption proved by the change in contour after recalcification.

Fig. 18. Case 6. History of parathyroid adenomectomy ten years prior to examination. Incompletely healed cyst-like lesion with dense sclerotic margin and bony septa in right superior pubic ramus. A similar lesion is discernible in right ilium. This patient also had persistent bilateral renal calcinosis and recurrent pyelonephritis. The skeleton was otherwise normal.

Fig. 19. Case 3. Staghorn calculus in left kidney and resorptive changes of lumbar spines.

Fig. 20. Case 4. Bilateral renal calcinosis and resorptive changes and deformity of lumbar spines.

evidence of bone disease has disappeared following parathyroidectomy. These lesions persist and resemble benign bone cysts with dense sclerotic margins and bony septa. Figure 18 shows such an incompletely healed cystic lesion in the superior pubic ramus ten years after parathyroidectomy in a forty-one year old female patient (Case 6). A similar lesion is present in the right iliac wing but more difficult to reproduce because of superimposed colonic gas and fecal shadows. The rest of the skeletal system and the blood chemistry were normal. Her renal calcinosis, however, persisted and she suffered from chronic and acute recurrent pyelonephritis.

THE RENAL CHANGES

In our small series of 8 cases, there are 6 patients with renal disease (4 with renal calculi and 2 with renal calcinosis, Fig. 9, 14, 19 and 20). All but one of the 6 also

Table VII

SUMMARY OF RENAL INVOLVEMENT

Case No.	Roentgenographic Findings	Chemical Findings	Other Findings
1	Normal	Normal	Normal
2	Renal calculi, bilateral, multiple	Elevated blood urea nitrogen, no acidosis	Chronic pyelonephritis
3	Renal calculi, left—staghorn, right—multiple	Elevated blood urea nitrogen, no acidosis	Chronic pyelonephritis
4	Renal calcinosis, bilateral	Elevated blood urea nitrogen, no acidosis	Chronic pyelonephritis
5	Renal calculus, left—solitary	Normal	Normal
6	Renal calcinosis, bilateral	Normal	Chronic and recurrent acute pyelonephritis
7	Renal calculi bilateral—recurrent	Elevated blood urea nitrogen, mild acidosis	Chronic and recurrent acute pyelonephritis
8	Normal	Normal	Normal

suffered from chronic pyelonephritis, and 4 patients showed chemical evidence of early renal functional impairment. These data are summarized in Table VII.

SUMMARY

Eight cases of primary hyperparathyroidism due to benign adenoma are reported.

The symptomatology, pathophysiology and chemical changes of primary hyperparathyroidism are briefly discussed.

The roentgenographic findings in osteitis fibrosa cystica generalizata are described:

1. The advanced changes are recapitulated in some detail with illustrative roentgenograms from 4 patients.

2. The less advanced bone changes are described with roentgenograms from one patient.

3. The very early bone changes are illustrated with roentgenograms of one patient.

4. The healing of osseous lesions after surgery is demonstrated by comparison of pre- and postoperative roentgenograms.

5. A partially healed cyst-like lesion ten years after parathyroidectomy is illustrated roentgenographically.

The renal complications are briefly described.

CONCLUSIONS

Primary hyperparathyroidism is a relentlessly progressive disease. The earlier the diagnosis is made and surgery carried out, the lesser is the damage to the skeletal and renal systems. Roentgenographically, osseous changes may be detectable in early stages of the disease. Such minimal abnormalities should arouse suspicion and lead to further diagnostic studies. Blood chemistry and certain newer techniques of laboratory examinatiom may be brought to bear on the problem until the diagnosis is established or excluded.

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THE SKULL IN HYPERPARATHYROID BONE DISEASE*

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ALTHOUGH cases of hyperparathyroidism with bone disease are not common, when encountered they may present a challenge to the diagnostic radiologist. The classic osseous changes of this disease are well known, and the often crucial diagnostic importance of subperiosteal bone resorption in the phalanges of the hands described by Camp and Ochsner[3,4] has been further documented by Pugh.[17,18] In the skull, however, particularly in the calvarium, the range and significance of the roentgenographic findings have not been so widely appreciated. Because of several recent experiences with unusual skull findings in cases of hyperparathyroidism, we were stimulated to review the roentgenographic findings in the 39 cases of proved primary hyperparathyroidism with osseous lesions seen at the Presbyterian Hospital (New York) in the years 1931 through 1958. A total of 70 cases of histologically verified primary hyperparathyroidism was seen during this period. Two of these cases were considered to have functioning parathyroid carcinomas, one with proved osseous metastases.

DESCRIPTION AND CLASSIFICATION OF PREOPERATIVE ROENTGENOGRAPHIC FINDINGS IN THE CALVARIUM

Since decalcification of bone especially marked subperiosteally is a major skeletal change in hyperparathyroidism with bone disease, it is not surprising that on roentgenograms the cortical bone and trabeculae become thin and indistinct. In consequence, the skull classically has a homogeneous ground glass-like appearance. In the calvarium the striking loss of bone detail is most evident at the cortical margins of vascular grooves and diploic venous channels and at the margins of the inner and outer tables (Fig. 1*A*).

As emphasized by Camp,[4] in many cases a miliary, mottled or granular pattern is visible in the bone of the calvarium (Fig. 2 *A*). On pathologic grounds, Jaffe[11] has suggested that this granular appearance is due to many thin, closely packed, partially calcified trabeculae of new bone which have formed in the fibrous tissue replacing the original bone. The granularity therefore

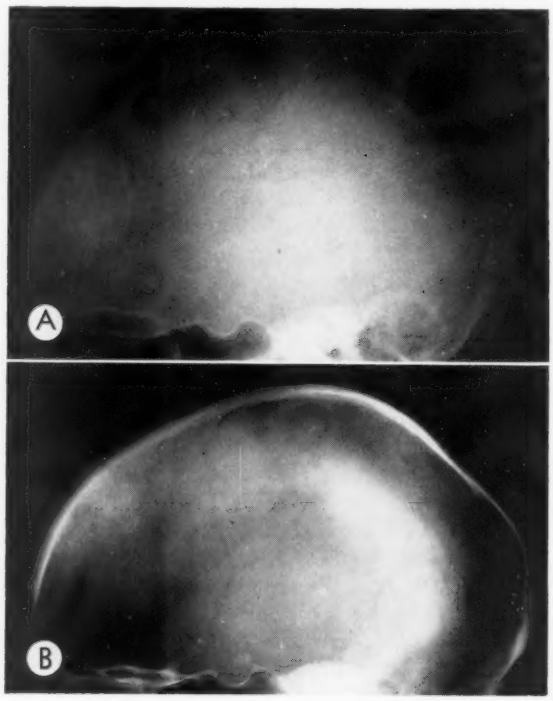

FIG. 1. P.H. 682482, forty-five year old woman with hyperparathyroidism. (*A*) August 6, 1942: Preoperative skull roentgenogram showing generalized ground glass type of appearance in calvarium (Class 1). (*B*) September 24, 1943: One year following removal of large parathyroid adenoma, skull has normal appearance. Note the reappearance of sharp outline to inner and outer tables and vascular grooves.

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represents relatively advanced complex bone disease.

Superimposed upon either the homogeneous ground glass-like or the granular mottled type of general alteration in the appearance of the calvarium, focal patches of bone resorption up to several centimeters in diameter may be seen (Fig. 3 *A*). These regions are often better appreciated in retrospect after some recalcification has occurred in the surrounding bone to provide improved contrast. The more gross bone expanding tumors or cysts relatively common in other bones in hyperparathyroidism, especially in the mandible and facial bones, are not commonly seen in the calvarium (one case in our series—Fig. 5, *A*, *B* and *C*).

Of great interest were the preoperative skull roentgenograms of 5 patients showing

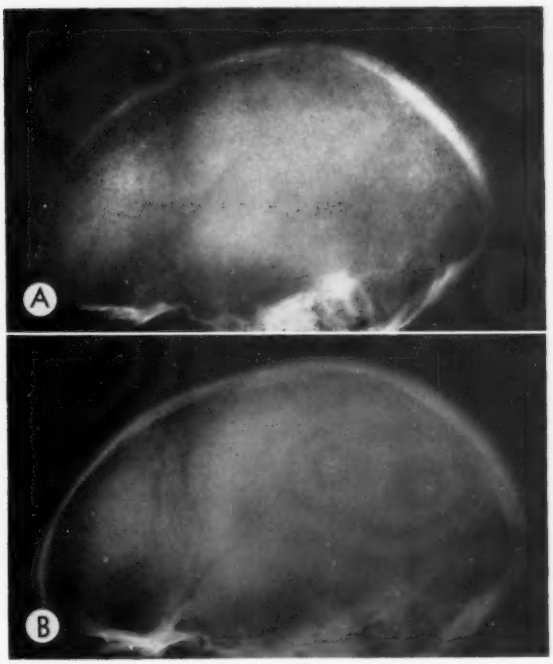


Fig. 2. P.H. 384613, thirty-four year old man with hyperparathyroidism. (*A*) July 13, 1933: Preoperative skull roentgenogram showing generalized granular or mottled type of alteration in the calvarium (Class II). (*B*) April 19, 1935: Almost two years after removal of a single parathyroid adenoma the skull has normal appearance. Note again the reconstitution of sharp margins to the inner and outer tables and the vascular grooves. The granular pattern is no longer present.

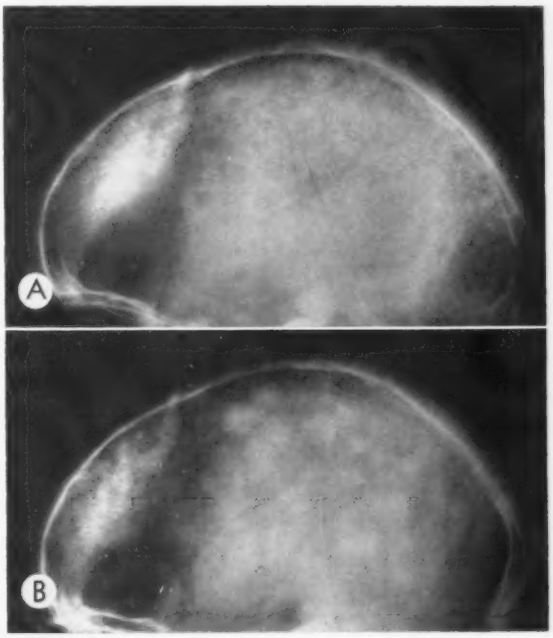


Fig. 3. P.H. 583499, forty-year old man with hyperparathyroidism. (*A*) July 14, 1939: Preoperative skull roentgenogram showing multiple areas of focal radiolucency in calvarium. Many of these regions are surrounded by a thin sclerotic margin. The intervening bone has granular texture (Class III). (*B*) September 16, 1958: Skull roentgenogram nineteen years following removal of a parathyroid adenoma shows dense sclerotic patches of bone apparently replacing the focal radiolucencies previously demonstrated. The intervening bone is normal.

focal or general sclerosis in the calvarium as the outstanding roentgenographic feature (Fig. 4 *A*). These cases are of special importance because of the possibility of diagnostic confusion with Paget's disease or osteoblastic metastases. And to make the differential diagnostic problem more difficult, thickening of the calvarium, so common in Paget's disease, has long been recognized as an occasional manifestation of hyperparathyroid bone disease. Since, however, the range of normal is not clearly defined for the various populations involved, how many of the reported cases have had thick skulls because of hyperparathyroid disease and how many represented chance associations cannot be stated with certainty. In none of these did the calvarium change in thickness on comparing

TABLE I

CLASSIFICATION OF THE ROENTGENOGRAPHIC FINDINGS IN THE CALVARIUM AS DEMONSTRATED ON PREOPERATIVE SKULL ROENTGENOGRAMS IN 30 CASES OF PRIMARY HYPERPARATHYROIDISM WITH BONE DISEASE

No. of Cases	Description of Findings
	Abnormal Calvarium
8	Class I. Generalized homogeneous, ground glass type of decrease in density of the calvarium, with loss of sharpness of cortical margins including the bony walls of vascular channels
10	Class II. Generalized mottled or coarsely granular texture of the calvarium with or without over-all loss of density but with loss of sharp cortical margins
5	Class III. Focal patches of decreased density superimposed upon a calvarium of Class I or II type or perhaps very rarely upon an otherwise normal appearing calvarium
5	Class IV. Focal patches of sclerosis in the calvarium superimposed upon any of the other classes or an otherwise normal appearing skull
28	
	Normal Calvarium
2	Normal skulls—despite definite subperiosteal resorption of bone of phalanges of hands in one case and bone resorption at ends of clavicles in the other
Total 30	

Note: Thickening of the calvarium and bone expanding tumorous lesions although mentioned in the text are not considered in this classification.

preoperation and postoperation skull roentgenograms.

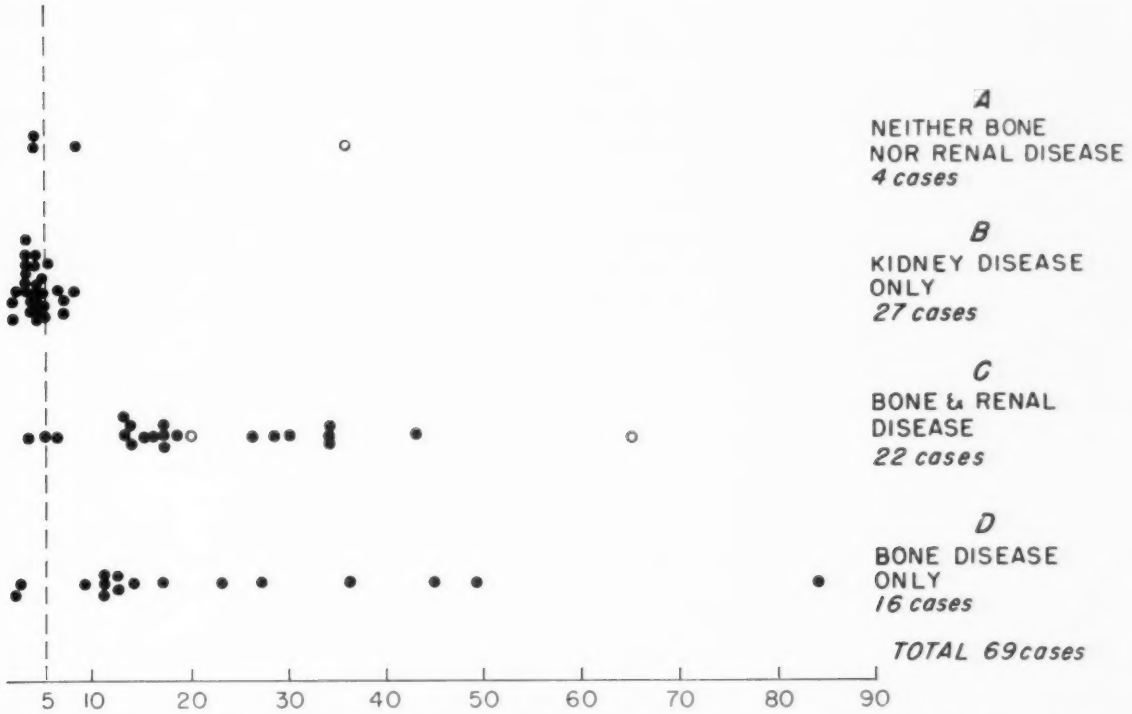
In accordance with the foregoing descriptions, the roentgenographic appearances of the calvarium in our cases are classified as shown in Table I. The borderlines, particularly between Classes I and II, are indistinct and arbitrary. Only 30 cases are included in the table since the preoperative skull roentgenograms in 7 cases were not available for review, although all had been considered to show evidence of hyperparathyroidism. In 2 additional cases no preoperative skull roentgenograms were made, but in each case impressive evidence of hyperparathyroid bone disease was visible elsewhere in the skeleton.

Upon review of the roentgenograms, we believe that in at least one-half and probably in two-thirds of our cases (16 of 23 cases in Classes I, II and III) the diagnosis of hyperparathyroid bone disease could be suggested on the basis of the preoperative skull roentgenograms alone. In retrospect, after comparisons with follow-up postoperative roentgenograms it could be seen that abnormalities had been present in all but 2 of these cases. In our experience, the distinction between the normal skull and abnormal skull of hyperparathyroid bone disease is not as easily appreciated as the distinction between the normal and the subperiosteally resorbed cortex of the phalanges of the hands in this disease. Nevertheless, in at least 3 of our cases the skull roentgenograms clearly indicated the possibility of hyperparathyroidism when the hands were normal.

No relationship between the appearance of the skull as classified in Table I and either the presence of renal calculi or the level of the preoperative serum calcium could be demonstrated.

CORRELATION OF BONE CHANGES WITH SERUM ALKALINE PHOSPHATASE

Preoperative serum alkaline phosphatase determinations were available in 69 of 70 patients with histologically verified para-



Graph 1. Preoperative serum alkaline phosphatase values (Bodansky units) in 69 cases of primary hyperparathyroidism—all histologically verified parathyroid adenomas, except for 2 functioning adenocarcinomas. The light circles represent patients with coexisting Paget's disease.

thyroid tumors. The distribution of these values based on the Albright and Reifenstein[1] classification is shown in Graph 1. Twenty-seven patients, with a history of renal calculi (Group B) and 4 patients with no history of renal calculi (Group A) showed no roentgen evidence of hyperparathyroid bone disease. Only 5 of the 31 patients in these two groups had preoperative alkaline phosphatase values higher than 5.0 Bodansky units (the upper limit of normal in our laboratory). Interestingly, the alkaline phosphatase values in these 5 patients returned to normal in the postoperative period. Also, a review of the skeletal roentgenograms of these 5 patients, including roentgenograms of the hands and skull, failed to show any evidence of bone involvement. It is probable that some patients with primary hyperparathyroidism have bone disease which is not sufficiently advanced to produce characteristic roentgenographic changes.[22]

Thirty-eight patients showed roentgen evidence of hyperparathyroid bone disease somewhere in the skeleton (Graph 1, Groups C and D). The preoperative serum alkaline phosphatase was significantly elevated in all but 5 of these patients. After resection of the parathyroid adenoma, the alkaline phosphatase gradually returned to normal in all except the 2 patients who had coexisting Paget's disease. Coincident with the decrease in serum phosphatase activity in the postoperative period, roentgenograms of the skeleton showed a gradual return toward normal. The 5 patients who had a normal or only slightly elevated preoperative alkaline phosphatase activity and roentgen evidence of skeletal involvement are of special interest. Four of these 5 patients had changes in the skull which were primarily sclerotic (Table 1, Class IV), and which were strikingly similar to the changes found on follow-up examination in 7 other cases after cure of the hyperparathyroidism.

It is well known that when the preopera-

tive serum alkaline phosphatase activity is normal or only slightly elevated in patients who have hyperparathyroidism, roentgen examination will usually fail to reveal any of the characteristic findings of hyperparathyroid bone disease. On the other hand, when roentgenograms of the skeleton and particularly of the skull show patches of bone sclerosis, a normal or only slightly elevated serum alkaline phosphatase determination does not exclude the possibility of hyperparathyroidism as the cause for these changes.

Findings in the Calvarium on Postoperative Follow-up Roentgenograms

Some insight into the reason for the variety of roentgenographic findings in the preoperative skull can be obtained by examination of the skull after surgical cure of the hyperparathyroidism.

On follow-up examinations in 7 of the 8 cases in Class I, those having a homogeneous ground glass type of appearance of the calvarium, 6 skulls returned to normal and in the other case patches of bone sclerosis developed as the calvarium was recalcified.

On follow-up examinations in 7 of the 10 cases in Class II, 4 skulls returned to a normal appearance whereas varying degrees of bone sclerosis developed in the calvarium in the other 3 cases. Two patients died of renal disease before definitive surgery was attempted, and the final patient in this group has had no follow-up skull roentgenograms.

Follow-up examinations in 4 of the 5 cases in Class III, cases having focal patches of radiolucency on preoperative roentgenograms, showed in 3 cases the development of sclerosis in the regions of former radiolucency (Fig. 3*B*), one of these ultimately showing evidence of recurrent hyperparathyroidism (Fig. 5, *B* and *C*). In the fourth patient the calvarium returned to normal after cure of the hyperparathyroidism. Thus, at least 7 of the 23 cases in Classes I, II and III developed sclerosis in the calvarium, coincident with healing of the bone lesions following cure of the hyperparathyroidism.

Finally, the follow-up examinations in the 5 patients in Class IV, cases having bone sclerosis in the calvarium on preoperative roentgenograms, revealed in 3 patients no essential change over periods of three to twelve years. (The patient without change for twelve years was originally reported by Gutman and Parsons[6] [Case 2.]) In another patient the focal patches of bone sclerosis became more dense and numerous (Fig. 4*B*), while in the fifth patient the skull ultimately returned to a normal appearance.

As has been shown above, focal sclerosis in the skull is often a manifestation of recalcification or healing. Since 4 of the 5 patients in Class IV had normal or near normal serum alkaline phosphatase levels and,

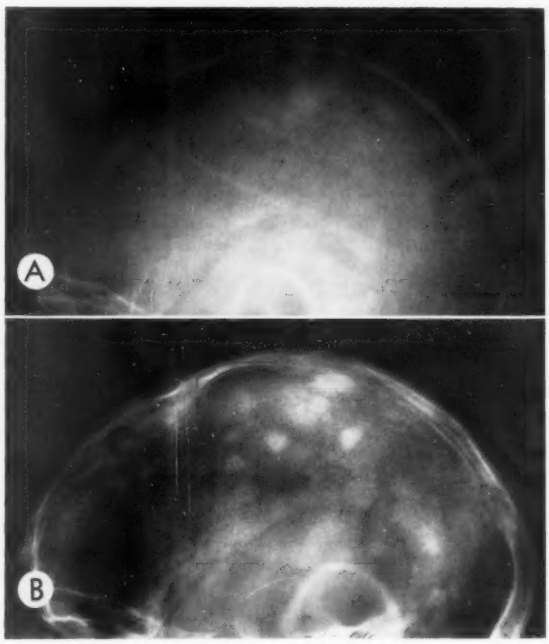


Fig. 4. Case 1. P.H. 841955, twenty-seven year old carpenter with hyperparathyroidism. (*A*) September 12, 1946: Preoperative skull roentgenogram showing multiple dense patches of sclerosis in calvarium. The linear streaks of increased density are artifacts (Class IV). (*B*) December 2, 1946: Three months following removal of parathyroid adenoma, the areas of increased density in the skull have become more numerous, discrete and dense. The intervening bone is normal.

in addition, showed persistent or even increased sclerosis of the calvarium following cure of their hyperparathyroidism, we believe that their preoperative sclerotic changes in the skull represent manifestations of partial or complete healing of hyperparathyroid bone disease.

DISCUSSION

In general, after surgical cure of hyperparathyroidism, the abnormal bone gradually over periods of weeks to months returns to a more normal appearance. The rate of this reconstruction seems to depend upon dietary intake of calcium, phosphorus and vitamin D, exercise and other factors. Return of sharply defined cortical bone density is the most characteristic feature of this healing process. Usually, the regions of focal bone destruction (including the bone expanding tumors) rapidly become densely calcified.[5] At times, this process may be so marked that the result can be mistaken for the surgical placement of bone chips in the osseous lesion. Fluid filled cysts may persist as sharply defined radiolucencies in the bone. On prolonged observation over periods of years, the bone gradually tends to be remodeled in the direction of normal.

As we have seen, the changes in the calvarium with healing are quite comparable to those which are described in the other bones. Sclerosis, focal or more general, has long been considered an occasional and expected response of the calvarium to cure of hyperparathyroidism.[17,21]

On the other hand, the development of focal or generalized osseous sclerosis in the skull, consistent with partial or complete healing of hyperparathyroid bone disease, prior to removal of a parathyroid adenoma has been described.[2,7,12–16,18,19,25 and others] Such sclerotic alterations in the calvarium were present in at least 5 of our 39 cases of primary hyperparathyroidism prior to surgical cure.

How is it possible that such changes may be found before operative cure of hyperparathyroidism? There are several possibilities. Changes in functional activity of parathyroid adenomas could result from local changes such as vascular thrombosis or hemorrhage leading to necrosis in the tumor.[8] Partial or complete healing of hyperparathyroid bone disease has been observed in a few patients following administration of large doses of substances with vitamin D activity, calcium and especially phosphorus, despite continuing chemical hyperparathyroidism.[13,22, our Case II] Of interest in this connection are Jaffe's[11] findings of bone sclerosis in rats maintained on high calcium diets while receiving long term injections of parathyroid extract despite evidences of rapid bone resorption in some parts of the skeleton. Of course, in practice, such treatment is recognized as hazardous because of the imminent danger of urinary tract calcification and secondary infection.

Since, as we have observed, patchy bone sclerosis in the calvarium may be the consequence of healing of the osseous lesions of hyperparathyroidism and since evidence of osseous healing has been known to occur in certain individuals prior to resection of a parathyroid adenoma, it seems quite possible that the bone sclerosis noted preoperatively in 5 of our patients may represent partial or complete healing of the hyperparathyroid bone disease in the skull. Follow-up observations support this interpretation of these cases. As previously mentioned, the fact that spontaneous healing did occur is further substantiated in 3 of these 5 patients by the presence of a normal alkaline phosphatase prior to surgery.

REPORT OF CASES

The 3 case reports to follow demonstrate the occurrence of focal patches of increased density in the calvarium in hyperparathyroidism. In the first 2 cases there is no doubt that the lesions are a consequence of hyperparathyroid bone disease. In the third case, we believe that the bone changes in the skull are a response to hyperparathyroidism, although the possibility of Paget's disease cannot be excluded.

Case I. P.H. 841955. W.S., a single white carpenter, first noted increasing weakness, loose teeth and symptoms of renal calculi in 1943 following an episode of pneumonia treated by sulfa drugs. When hospitalized elsewhere in 1944 because of left ureteral calculi, the serum calcium was 14.7 mg. per cent, phosphorus 3.4 mg. per cent, and alkaline phosphatase 16.6 Bodansky units. Weakness and episodes of flank pain continued until August, 1946, when a pathologic fracture through a cystic lesion in the right femoral shaft was sustained. Treatment with bilateral hip spica and bed rest was accompanied by severe nausea and vomiting.

When first admitted to this hospital in September, 1946, roentgenographic examination showed some general demineralization of bone with subperiosteal bone resorption in the phalanges of the hands and in the femora. Several cyst-like lesions were noted in the long bones. Peculiar patches of sclerosis were seen in the skull (Fig. 4 *A*). Left renal calculi were demonstrated. The serum calcium was 20.3 mg. per cent, serum inorganic phosphorus 2.9 mg. per cent, alkaline phosphatase 6.8 Bodansky units, and urea nitrogen 14 mg. per cent.

On September 28, 1946, at the age of twenty-seven years, a 6 cm. diameter parathyroid adenoma was removed from the intracapsular portion of the upper pole of the left lobe of the thyroid. In addition, a well differentiated papillary carcinoma was excised from the right thyroid lobe. Postoperatively, the serum alkaline phosphatase rose to 10.5 Bodansky units, while the serum calcium dropped as low as 8.0 mg. per cent. Three months later the multiple patches of increased density in the skull were more numerous, discrete and dense (Fig. 4 *B*). Roentgenograms of the other bones showed evidence of healing of the hyperparathyroid bone disease. Except for refracture of the right femur three months postoperatively, recovery was uneventful and the patient was well in 1957.

Case II. P.H. 868500. N.M., a forty-two year old woman, was first admitted in 1947 with a chief complaint of multiple bone tumors of two years' duration. She had been well until a pathologic fracture of the right forearm was sustained in 1944. At that time the bones, in general, were found to be decalcified. Soon after this, right renal colic was first experienced. Two years prior to admission pain and marked swelling over the outer end of the left clavicle first appeared, which was followed by development of similar tumors over the left iliac crest and left jaw. Roentgenographic examination demonstrated bone destruction in these regions as well as in multiple other sites. For some months prior to admission the patient had been treated with large doses of calcium ascorbate, calcium gluconate and viosterol. With this treatment she continued to lose weight and have bone pain, although her physician felt that the bone tumors were becoming smaller.

On admission, roentgenographic examination of the skeleton showed many cystic lesions including large cystic bone tumors which had broken into the soft tissues at multiple sites including those mentioned above. A comparison with previous roentgenograms showed that many of these tumors had become partially calcified, apparently as a response to the high calcium and vitamin D intake. Interestingly, there was no subperiosteal bone resorption in the phalanges and the serum alkaline phosphatase was only 5.4 Bodansky units. The serum calcium was 17.9 mg. per cent and the serum phosphorus was 3 mg. per cent. The skull showed loss of trabecular sharpness and multiple regions of radiolucency. Many of the radiolucent areas had sclerotic margins (Fig. 5*A*). Multiple right renal and ureteral calculi were present and the blood urea nitrogen was 19 mg. per cent. While in the hospital a pathologic fracture of the left femoral neck occurred. Biopsy of the mandible revealed osteitis fibrosa.

On June 4, 1947, a parathyroid adenoma weighing 4.7 gm. was removed from behind the lower pole of the left lobe of the thyroid. Postoperatively the serum calcium fell to a low of 8.5 mg. per cent, and the alkaline phosphatase rose to 16.5 Bodansky units, then gradually returned to normal. Rapid clinical and roentgenographic improvement in the bone disease occurred, but the patient remained bedfast several months because of the left femoral fracture, finally making an excellent clinical recovery with return to limited activity.

Multiple right renal and ureteral calculi and a nonfunctioning right kidney led to a right nephrectomy in 1949. Nevertheless, low grade elevation of the blood urea nitrogen and low grade urinary tract infection persisted. Subsequent to 1949, the serum calcium varied between 10 and 12.5 mg. per cent and the serum phosphorus ranged from 3.6 to 4.7 mg. per cent.

Roentgenograms of the skull eight years following removal of the parathyroid adenoma

showed development of multiple patches of bone sclerosis in former regions of radiolucency (Fig. 5*B*).

The patient continued feeling well until 1957, when insidious anorexia, weight loss and weakness began. In the fall of 1958 she began to have severe weakness and pains in the hips and legs. The alkaline phosphatase was found elevated to about 13 Bodansky units. Skeletal survey examination showed recurrence of bone demineralization generally, including subperiosteal resorption in the hands and resorption of the distal ends of the clavicles. A remarkable change had occurred in the skull. In place of the focal areas of bone sclerosis which had appeared in response to the cure of the original hyperparathyroid bone disease, there were focal areas of radiolucency (Fig. 5*C*). The blood urea nitrogen was 44 mg. per cent, serum calcium 11.5 mg. per cent, serum phosphorus 5.3 mg. per cent. Based upon creatinine clearance, tubular resorption of phosphate was 47 per cent (normal, 78–94 per cent).

On January 13, 1959, repeat neck exploration was performed with removal of five additional masses of parathyroid tissue which were interpreted histologically as representing hyperplasia, probably secondary.

The serum calcium fell to a low of 9.9 mg. per cent and no evidence of tetany developed. The patient recovered from the operation and was returned home.

Case III. P.H. 583808. B.S., a forty-six year old single woman garment worker, was seen in 1956 because of upper abdominal distress, headaches, two years of generalized aching and joint stiffness, as well as increasing lassitude. Physical examination was unremarkable except for bilateral nodular thyroid. Serum calcium was 14.8 mg. per cent, inorganic phosphorus 2.6 mg. per cent, alkaline phosphatase 4.6 Bodansky units, and blood urea nitrogen 9 mg. per cent. Skeletal roentgenograms showed scattered areas of bone sclerosis in the lumbar spine, femora and pelvis, and patches of bone sclerosis in a relatively thick skull, resembling the changes not infrequently seen in other patients after regression of the bone disease following cure of hyperparathyroidism (Fig. 6).

In 1939, she had had a partial thyroidectomy for a nontoxic nodular goiter, including a 5 cm. ovoid cystic mass at the lower pole of the left lobe of the thyroid. For several months before this operation she had noted weakness, headaches and an enlarging lump in the left thyroid region. At that time skull roentgenograms were reported as showing a relatively thick calvarium. Postoperative muscle twitching and headaches were treated with various agents, including calcium gluconate. Cholecystectomy, hys-

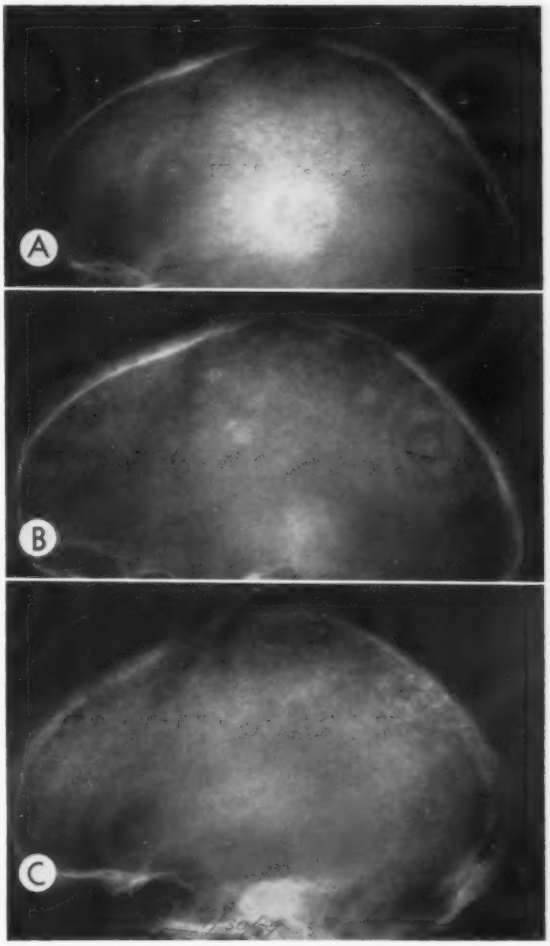


Fig. 5. Case II. P.H. 868500, forty-two year old single female with hyperparathyroidism. (*A*) May 12, 1947: Preoperative skull roentgenogram showing general loss of sharpness of cortical bone and multiple focal regions of radiolucency, many with dense margins, including a particularly large lesion in the inferior parietal region which on other views not shown here is seen to bulge into the middle cranial fossa. (*B*) September 7, 1955: Skull roentgenogram eight years following removal of a large parathyroid adenoma showing multiple patches of bone sclerosis replacing former regions of radiolucency. (*C*) January 3, 1959: Skull roentgenogram three years and four months later when patient had developed recurrent hyperparathyroidism resembles that originally seen (*A*) except that focal areas of radiolucency are more numerous. Note that the patches of increased bone density have been partially resorbed.

Fig. 6. Case III. P.H. 583808, forty-six year old single woman with hyperparathyroidism. September 21, 1955: Preoperative roentgenogram shows a relatively thick skull with numerous and confluent patches of increased density. Follow-up examination three years after operative cure of hyperparathyroidism showed no change.

terectomy, and abdominal exploration for pancreatitis were performed in the years 1946 to 1953. In 1955, roentgenograms of the skull had demonstrated patches of sclerosis in a thick skull, at first considered to represent Paget's disease, but identical to findings on later examinations.

On November 15, 1956, a 16×5×3 mm. parathyroid adenoma was excised from the region caudad to the lower pole of the left lobe of the thyroid. A partial thyroidectomy for nontoxic nodular goiter was also performed. Following operation the serum calcium fell slowly to 10.2 mg. per cent. The patient has remained well to 1959 with no roentgenographic change in the appearance of the skull.

DIFFERENTIAL DIAGNOSIS

The generally decalcified skull of the ground glass or mottled granular types of Classes I and II is very nearly specific for primary or secondary hyperparathyroidism. The major problem, as already mentioned, is to distinguish the abnormal from the normal calvarium. Once the skull is recognized as being abnormal, having the characteristics described above, hyperparathyroid bone disease is the most likely diagnosis. Further support for this diagnosis, however, should be obtained by serum calcium, serum phosphorus and serum alkaline phosphatase determinations, and by skeletal survey examination with special attention to the hands, the ends of the clavicles and, in younger individuals, the lamina dura. Distinction between primary hyperparathyroidism and renal osteodystrophy (with secondary hyperparathyroidism) cannot be made on the basis of skull roentgenograms.

Differential considerations include certain cases of osteomalacia[10] as well as the osteoporosis of Cushing's disease[9,24] which have been reported sometimes to produce similar findings in the skull. Occasionally, in the aged, senile osteoporosis may be confused roentgenographically with hyperparathyroid bone disease. In children, in whom primary hyperparathyroidism is very rare, neuroblastoma, leukemia or the severe hereditary anemias may produce changes in the calvarium resembling hyperparathyroid bone disease. Very rarely metastatic carcinoma, multiple myeloma, radium poisoning, and hypervitaminosis D might produce similar appearing changes in the calvarium.[1] Fortunately, the characteristic blood chemical findings, the changes in other bones and the clinical history will nearly always obviously distinguish cases of hyperparathyroid bone disease.

When focal patches of bone resorption are also present as in Class III, the differential considerations become wider. Usually, however, in these cases the characteristic granular or mottled pattern in the bone surrounding the focal regions of bone resorption will suggest the correct diagnosis. In children particularly, the reticuloses, neuroblastoma and other rarer metastatic tumors would have to be considered. In adults, multiple myeloma or metastatic breast and lung carcinoma or other neoplasm should merit diagnostic consideration. Osteomyelitis of various origins may produce similar findings. Very rarely such appearances have been described in osteomalacia (possibly related to compensatory parathyroid overactivity).[10]

Again, these considerations are usually ruled out by the clinical history and blood chemistry findings. Because of the many disorders, including multiple myeloma and metastatic carcinoma, which may be associated with hypercalcemia, correct interpretation of this blood chemical finding may be very difficult.[23]

Finally, in the Class IV type of calvarium with focal patches of bone sclerosis, the surrounding bone is typically normal in appearance and the diagnosis may be most difficult (Fig. 4–6). Paget's disease of the skull as well as osteoblastic metastases may exactly mimic these findings. We believe with Gutman and Parsons[6] and others[1,20,21] that any patient suspected of having Paget's disease on the basis of skull roentgenograms of this type should have serum calcium, serum phosphorus and serum alkaline phosphatase determinations before ruling out hyperparathyroidism.

SUMMARY

The roentgenographic findings in the calvarium of 39 patients with primary hyperparathyroidism and bone lesions have been reviewed and classified.

Typically, the calvarium has characteristic homogeneous ground glass or mottled granular patterns of decreased density. Occasionally, focal patches of bone resorption are present, making the differential diagnosis more difficult. In a few cases focal patches of bone sclerosis are seen in the calvarium of patients with active hyperparathyroidism. Circumstantial evidence is presented which indicates that such changes are the consequence of partial or complete healing of previously active bone disease. In these cases, distinction from Paget's disease or osteoblastic metastases may be extremely difficult.

In two-thirds of these patients, the diagnosis of hyperparathyroid bone disease could be suggested on the basis of the skull roentgenograms alone. In retrospect, nearly all cases showed some involvement of the calvarium. Roentgenograms of the skull were often of positive value in making the correct diagnosis.

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ADVANCED SKELETAL CHANGES IN HYPERPARATHYROIDISM

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NUMEROUS articles have appeared in the world medical literature in the past years defining the clinical, biochemical, and roentgenographic appearance of hyperparathyroidism. It has been stated that the diagnosis of hyperparathyroidism is usually based upon the findings of either renal calculi, renal calcification or a peculiar but definite abnormality of the bones.

Several authors have emphasized that approximately one-third of the cases of proved hyperparathyroidism will present with diffuse osteoporosis, one-third will present with definite skeletal findings, and one-third with no visible roentgenographic changes in the skeletal structures. We have encountered 3 advanced cases which grossly outline the marked changes which may occur in primary and secondary hyperparathyroidism. We are presenting these case histories to emphasize the important skeletal changes of this disease.

The cases of hyperparathyroidism which demonstrate skeletal changes usually show one or more of the following: (a) diffuse osteoporosis usually associated with a granular mottling of the skull and to a lesser extent of the long bones; (b) subperiosteal bone resorption most often seen in the fingers, clavicles and medial aspects of the tibia; (c) cystic changes in the bone which are in reality solid tumors of low density composed of osteoblasts and osteoclasts and are the end results of increased metaplasia of bone; and (d) periarticular calcifications often localized in areas of trauma and often associated with pathologic fractures.

The following cases demonstrate to an advanced degree all of the skeletal changes enumerated above.

REPORT OF CASES

CASE 1. S.S., a thirty-three year old white female, was first seen in May, 1951 with headaches, weakness, and dizziness of several years' duration. Her laboratory data revealed a urea-nitrogen of 70 mg., phosphorus of 5.3 mg., creatinine of 6 mg., and hemoglobin of 7.7 gm. The diagnosis was chronic pyelonephritis with chronic anemia and azotemia. One month later skeletal changes consistent with secondary hyperparathyroidism were recognized.

In 1952 and 1953, the calcium level varied between 11.1 and 12 mg., and the phosphorus level between 5.8 and 8.3 mg. The alkaline phosphatase was 24.8 Bodansky units, and the Sulkowitch test was positive. Bone marrow aspiration revealed changes consistent with osteitis fibrosa cystica. On the final admission, the blood urea nitrogen was 140 mg., the creatinine 9.5 mg. The patient expired from uremia with terminal bronchopneumonia.

Autopsy revealed bilateral contracted kidneys due to long standing pyelonephritis. The parathyroids were hyperplastic but a true adenoma was not found.

The roentgenographic findings in the later stages of the disease are classic of hyperparathyroidism. The skull and mandible (Fig. 1) reveal the typical granular mottled osteoporosis seen in hyperparathyroidism with lack of detail of both the inner and outer tables. The probability of small cystic areas within the calvarium is suggested by the areas of increased radiolucency. The absence of the lamina dura is well shown in the teeth arising in the mandible. The frontal roentgenogram of both shoulders (Fig. 2) reveals marked subperiosteal resorption of the distal fourth of the clavicles. Soft tissue calcification is present bilaterally, primarily in the region of the acromioclavicular joints. Roentgenogram of the left shoulder (Fig. 3), taken approximately three months prior to the patient's demise and eight months after the previous roentgenograms, reveals the extensive periarticular calcification presenting as large

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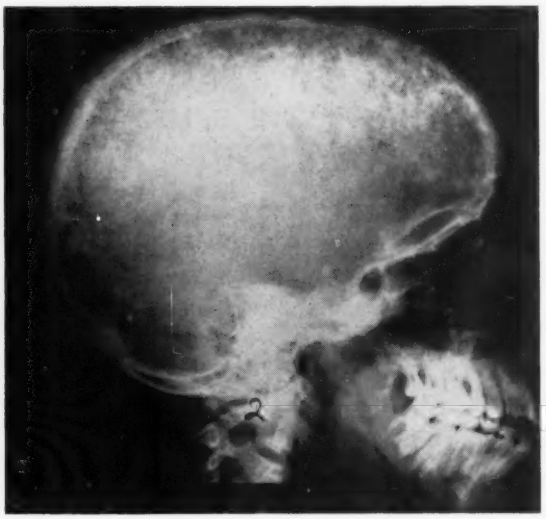

Fig. 1. Case 1. Roentgenogram of the skull shows granular mottled osteoporosis with lack of detail of the outer and inner tables, areas of increased radiolucency in the calvarium and absence of the lamina dura of the teeth arising in the lower jaw.

masses in the soft tissues of the shoulders. At this time, approximately half of the clavicles had been resorbed. Marked periosteal resorption of bone is also noted along the medial margin of the humerus together with a granular osteoporosis of the shaft associated with cortical and medullary cystic changes. Similar and even more marked subperiosteal resorption is demonstrated in a roentgenogram of the hand (Fig. 4) which shows the typical lace-like pattern, most marked in the shafts of the middle phalanges but present throughout all of the visualized bones. Again, the extensive decalcification and granular osteoporosis are observed. As noted in

Fig. 2. Case 1. Roentgenogram of both shoulders demonstrates marked subperiosteal resorption of the distal ends of the clavicles with soft tissue calcification in the region of the acromioclavicular joints.

Fig. 3. Case 1. Roentgenogram of the left shoulder eight months later shows marked advance, especially in the periarticular calcification.

many of the advanced cases, pronounced resorption of the tufts of the distal phalanges is

Fig. 4. Case 1. Roentgenogram of the hand reveals marked subperiosteal resorption with a lace-like pattern, particularly pronounced in the shafts of the middle phalanges, and granular osteoporosis.

Fig. 5. Case I. Roentgenogram of the pelvis in advanced stage of the disease reveals intracapsular fractures of both femoral necks with resorption of the fragments, marked osteoporosis, soft tissue calcification and sclerosis of the blood vessels.

present. Vascular calcifications are present but there is no evidence of periarticular calcification. Roentgenograms of the pelvis taken in January, 1952 revealed marked decalcification of the pelvis and femora without evidence of periarticular calcification. The roentgenogram taken in May, 1953 (Fig. 5) reveals that bilateral intracapsular fractures of the femoral necks had occurred in the interval with increasing osteoporosis and marked resorption of the skeletal fragments. Soft tissue calcification had occurred at the fracture site together with vascular calcification of the iliac and femoral vessels.

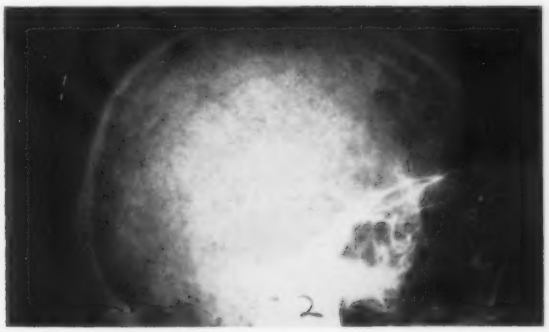

Fig. 6. Case II. Roentgenogram of the skull shows changes similar to those seen in Figure 1.

Case II. K.J., a twenty-five year old white female, entered the hospital in 1947 with a three month history of increasing fatigue, anorexia and listlessness. Physical examination revealed a thin, nervous young woman with slight tremor of the tongue and hands. The blood pressure was 206/110. A firm mass of the lower neck was considered to be an enlarged thyroid. The basal metabolic rate varied from 5 to 13 with a cholesterol of 227 mg. The nonprotein nitrogen was 37 mg., hemoglobin 13.6 gm. The patient was treated for a possible thyroiditis. In November, 1952, the patient was re-admitted because of ankle edema, intermittent fever to 101° F. and 18 pound weight loss. Pertinent laboratory findings revealed a hemoglobin of 9 gm., urea 19 mg., serum calcium 17.6 mg., phosphorus 3.3 mg. and 2.9 mg., alkaline phosphatase 25.7 Bodansky units, and a

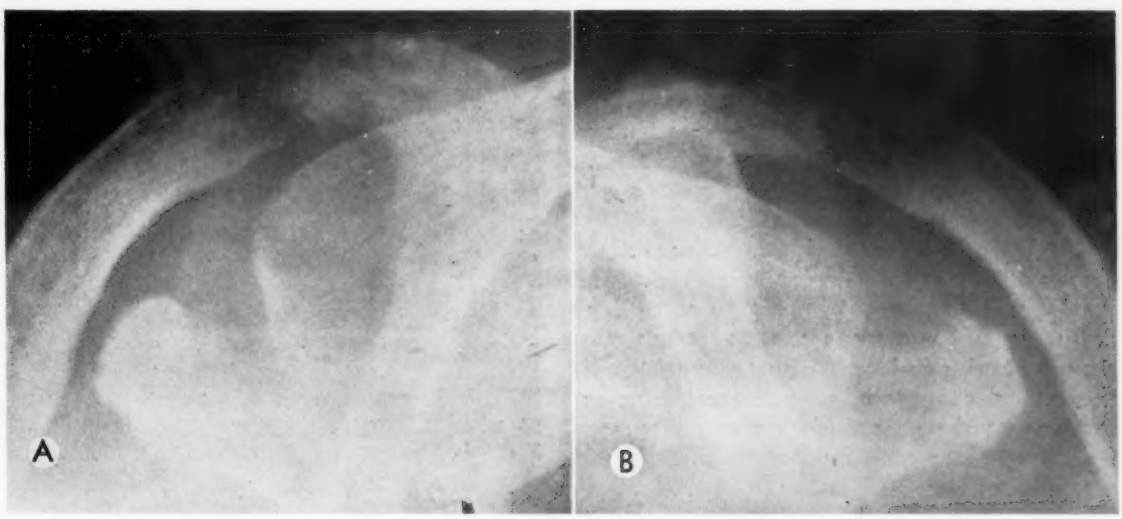

Fig. 7. Case II. (*A*) Left shoulder. (*B*) Right shoulder. There is subperiosteal bone resorption at the acromioclavicular joints with marked peripheral osteoporosis and loss of definition of the distal ends of the clavicles.

Fig. 8. Case II. Roentgenogram of the hand shows changes similar to those seen in Figure 4 but of a lesser degree.

Sulkowitch test of 2+. The patient's course was rapidly downhill and she expired in December, 1952.

Autopsy revealed a parathyroid adenoma 1.5 cm. in diameter near the lower border of the right thyroid. The three other parathyroids were normal. Diffuse calcification was present throughout the kidneys, the mucosa of the gastrointestinal tract and many other organs as well as the synovia around many of the articular surfaces. Marked decalcification was noted in all of the long bones and skull.

The roentgenographic findings are again characteristic of hyperparathyroidism. The roentgenogram of the skull (Fig. 6) reveals the diffuse granular osteoporosis and roentgenograms of both shoulders (Fig. 7, *A* and *B*) show the typical changes of subperiosteal bone resorption at the acromioclavicular joints with marked peripheral osteoporosis and loss of definition of the distal ends of the clavicles. A roentgenogram of the hand (Fig. 8) demonstrates irregular areas of osteoporosis with subperiosteal bone resorption, most marked along the lateral margins of the middle and proximal phalanges. The characteristic lace-like appearance of the cortex is again well visualized. Roentgenogram of the right knee (Fig. 9) reveals fluid in the suprapatellar pouch with calcification of the synovia. Marginal bone resorption and cyst formation were present in many of the long bones as shown in the femur and tibia (Fig. 9).

Case III. E.B., a twenty-year old female, was admitted to the hospital because of fracture of the left femur. The laboratory findings revealed the calcium level to vary between 10 and 12 mg. and the phosphorus between 4 and 7 mg.

Roentgenograms of the long bones revealed typical changes, such as those shown in Figure 10, consisting of expanded cystic areas in the shafts of the bone with thinning of the cortex and a rather sharp demarcation of the cystic region from the normal bone. The uninvolved bones, such as the tibia shown in the illustration, appeared normal. The left femur (Fig. 11)

Fig. 9. Case II. Roentgenogram of the right knee demonstrates fluid in the suprapatellar pouch with calcification of the synovia.

Fig. 10. Case III. Roentgenogram of the bones of the leg shows expanding cystic areas with thinning of the cortex of the fibula. The tibia is normal.

Fig. 11. Case III. Roentgenogram of the upper two-thirds of the left femur reveals a pathologic fracture associated with cystic changes in the femur, ischium and pubic bone.

had a pathologic fracture in the mid-third of the shaft associated with numerous cystic changes in the neck and head as well as in the pelvis, including the ischium and pubis.

A parathyroid adenoma was removed surgically.

SUMMARY

The skeletal changes in hyperparathyroidism may be summarized as follows: (1) diffuse osteoporosis; (2) subperiosteal bone resorption; (3) cystic appearing lesions of bone; and (4) periarticular calcifications.

Due to early medical diagnosis, the radiologist rarely encounters advanced skeletal lesions of hyperparathyroidism in modern practice. We have presented 3 such cases to re-emphasize the characteristic changes seen in this disease.

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ROENTGEN ASPECTS OF TIETZE'S SYNDROME*

PAINFUL HYPERTROPHY OF COSTAL CARTILAGE AND BONE—OSTEOCHONDRITIS?

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TIETZE'S syndrome consists of painful, nonsuppurative swelling of one or more costal cartilages and bone, and is of unknown etiology. The process is benign and self limited, but often leaves residual tumorous deformities of the affected areas. It was first described by Tietze in 1921,[20] and slowly received recognition in Europe and Japan.[18] It was not until 1953[16] that the first cases were reported in the American literature, and since then about 20 additional cases have been described.[1,7,8,10,11,16,19,21] A total of 200 cases has now been reported, but most observers agree that the incidence of the disease is much greater than this figure might indicate. The lesions may go unrecognized or unrecorded, or they may be classified as osteochondritis, "costochondral" syndrome, or as miscellaneous chest disorders. Physicians in a large midwest clinic, alerted to the syndrome, found 13 cases in a two year period.[10] In a military installation, 22 cases were detected in one year.[8]

It is important that this syndrome be recognized in order to differentiate it from more serious causes of chest pain. It is likely that any patient with chest pain these days will have a chest roentgenographic examination. The radiologist then has an excellent opportunity to help establish an early diagnosis by being aware of the syndrome and by recognizing the roentgen and clinical signs which may appear.

A résumé of the clinical features may be presented as follows. There is an insidious or acute onset of pain and swelling over one or more costal cartilages. Usually the costochondral or the chondrosternal junctions are involved. The pain may precede or follow the development of a tumor-like swelling, or they may appear simultaneously. The pain is accentuated by cough, motion, and deep respirations. It may radiate laterally and to the arm. The swelling, or mass, is of firm consistency and is tender for a variable duration of time. The overlying skin is normal and freely movable. The second costal cartilage is most frequently involved. According to Kayser[11] about 69 per cent of these patients have only a single swelling occurring with equal frequency on each side. Thirty-one per cent have multiple sites involved; of these, a considerable number have three or more lesions. There is no lymphadenopathy, and it is generally believed that no febrile response takes place. The laboratory studies in uncomplicated cases are entirely normal. If left to run its course, the tenderness and pain gradually disappear and the swelling regresses slightly over weeks and months, but a slightly protruding chest deformity usually remains. The sexes are equally affected. The ages of the patients range from eleven to seventy-nine years with a median of twenty-eight years.[11] There is no correlation with the type of occupation. For detailed compilations and analyses of clinical findings in all of the previously published cases, the reader is referred to the publications of Kayser[11] and Karon *et al.*[10]

The roentgenologic aspects have not been previously demonstrated or adequately discussed in works of American origin, or in any of the commonly available European publications. The primary purpose of this communication is to present 2 cases of Tietze's syndrome, in 1 of which marked skeletal changes were seen to develop and progress over an eight year

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period. The microscopic pathology will be correlated with the roentgen findings. The second case presents comparatively minimal roentgenographic and biopsy abnormalities.

REPORT OF CASES

Case 1. First Admission. A.G., a twenty-five year old white male, was admitted to the hospital in October, 1949, with a six week's history of aching and stiffness of his shoulders, and pain in the region of the chondrocostal junctions, bilaterally. There was no joint swelling, erythema, or tenderness until one week before admission, when his left sternoclavicular joint became swollen and tender. There was no history of trauma. He had lost an undetermined amount of weight with this illness.

Physical examination. An indefinite area of tenderness and induration adjacent to the left sternoclavicular joint was noted. Another tender swelling was present along the left fifth rib in the area of the costochondral junction. Scattered skin lesions, some pustular and some crusted, each about 2 mm. in diameter, were present on the hands, feet, thighs and legs.

Laboratory data. The white blood cell count varied from 4,000 to 14,000. The differential count was normal on most occasions. The urine was negative. Bence Jones protein was absent on several tests. The blood sedimentation rate was 32 mm. in one hour. Total serum protein was 7.7 gm. per cent, with 2.38 gm. albumin and 5.32 gm. globulin. Tuberculin skin test was negative. Serologic test for syphilis was negative. Cultures of the blood and skin were negative as were a series of agglutination tests for *Brucella* and other enteric pathogens.

Course. The patient's temperature fluctuated from normal to 100° F. The pain in the rib areas and chondrosternal junctions on the left subsided gradually but some swelling remained. Rather suddenly, tenderness developed over the right first rib at the costochondral margin. This receded gradually over a period of two to three weeks. Several days after admission, neurologic disorders appeared and in twelve hours the patient developed almost a total paralysis. Studies of bone marrow, liver, and muscle tissue were all normal. A biopsy of the mass over the first costochondral junction was performed. This showed collections of mononuclear cells adjacent to irregular bone trabeculae and was interpreted as subacute osteochondritis (Fig. 5). With physiotherapy, he slowly recovered about 80 per cent of normal muscular function. His skin disease lasted almost a year before disappearing. The mass in the chest wall remained.

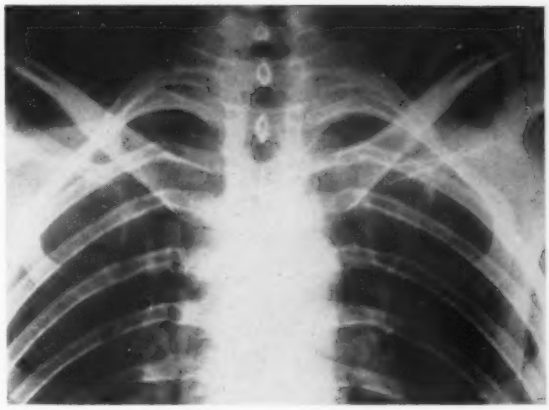

Fig. 1. Case 1. October 31, 1949: The anterior portions of the first ribs are expanded and show an irregular calcified or ossified trabecular pattern.

Roentgen findings. A chest roentgenogram on admission, October 31, 1949, revealed expansion of the anterior portions of both first ribs extending to the angle of the ribs laterally (Fig. 1). Irregular trabeculations were present within this abnormally splayed section of ribs. The other ribs appeared normal. The sternoclavicular joints were also normal in special studies. Lateral roentgenograms showed no retrosternal masses. Repeat examination on November 30, 1949, showed increased density of the enlarged anterior sections of the first ribs, bilaterally, due to deposition of calcium or bone (Fig. 2, *A* and *B*). Proximal to the expanded anterior segment of the right first rib, periosteal new bone was seen, and a similar layer of new periosteal bone had appeared about the superior margin of the right second rib anteriorly. In addition, the anterior segment of the left third, fifth and sixth ribs had undergone a loss of bone density for varying distances from the costochondral junction as compared to the right side and to previous roentgenograms. Examination on January 6, 1950, showed considerable increase in calcium and bone density of the anterior portions of both first ribs (Fig. 3, *A* and *B*). The new periosteal bone about the second rib on the right was being incorporated into the cortex of the bone. The roentgenologic deduction from the sequence of examinations was that the process was an unusual form of osteochondritis of unknown etiology.

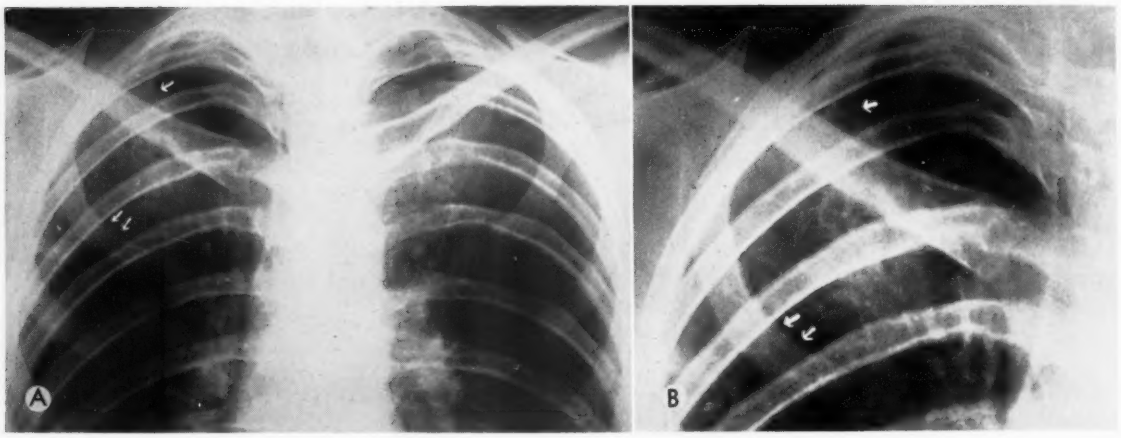

Fig. 2. Case 1. November 30, 1949: (*A*) Roentgenogram made one month after Figure 1 shows increased density of the enlarged anterior sections of the first ribs bilaterally, due to deposition of calcium or bone. Periosteal new bone (arrows) is present about the right first rib and is seen above the superior margin of the second right rib anteriorly. (*B*) Detail of right upper ribs to better delineate the expansion of the first rib and periosteal new bone of the first and second ribs (arrows).

Subsequent course. Following discharge, the patient later in 1950 underwent an appendectomy in another hospital. In 1951 he developed signs and symptoms of hyperthyroidism, which was treated with I^{131} and later a subtotal thyroidectomy was performed with good results. He had episodes of severe pain in the left upper chest in 1953, and again in 1957, relieved on each occasion by aspirin. In May of 1957, he had recurrent thyroid hyperactivity which was controled by I^{131} therapy.

Second Admission. In September, 1958, the patient was re-admitted with a history of several months of intermittent and variable neurologic disturbances including abnormalities in gait and vision, paresthesias, loss of the sense of touch, and pain.

Physical examination. There were many neurologic deficits. There was a hard nontender enlargement of both sternoclavicular joints, more marked on the left.

Laboratory data. The white blood cell count was 9,850, with a normal differential. The hematocrit was 48 per cent. Tests of urine showed 2 to 8 white cells per high power field, and were twice negative for porphyrins. Urine calcium in a twenty-four hour specimen was normal. Total serum proteins were 8.27 gm. per

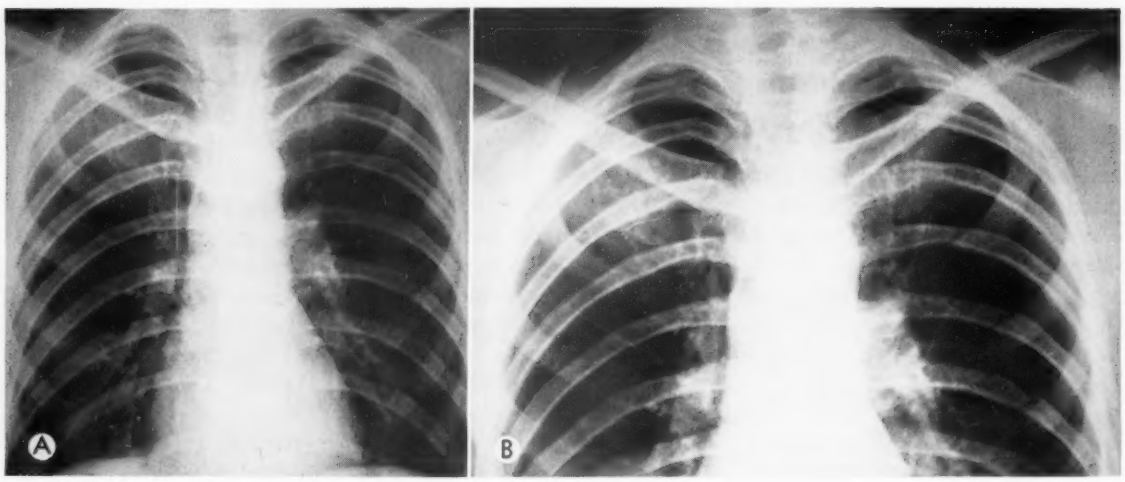

Fig. 3. Case 1. January 6, 1950: (*A*) A little more than three months after initial roentgenogram there is further increase in calcification and ossification of the first ribs. Periosteal new bone is being incorporated into the rib structures. (*B*) Detail showing the anterior ribs and periosteal changes.

Fig. 4. Case 1. June 7, 1958: Roentgenogram taken approximately eight and one-half years after initial roentgenogram. The first ribs are markedly enlarged and dense. The entire left first rib and most of the right first rib are involved. Note also increased density of the anterior portions of the fifth, sixth, and seventh ribs on the left as compared to the right.

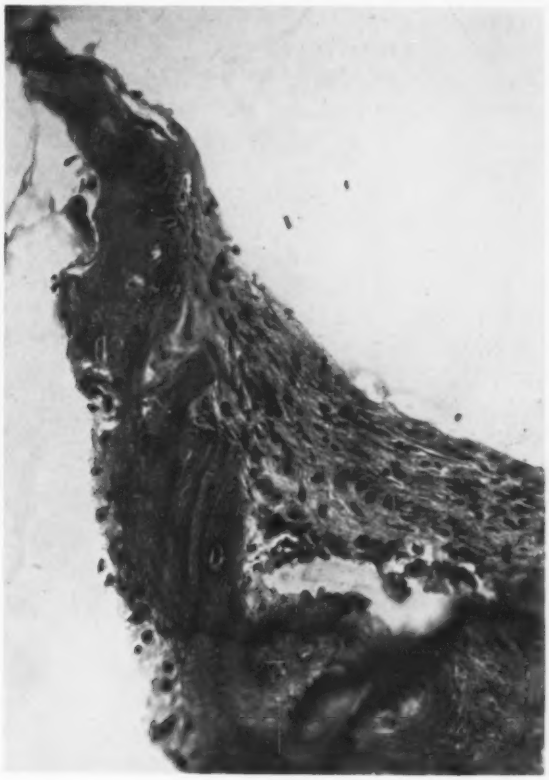

Fig. 5. Case 1. Biopsy specimen of left first costochondral mass shows mononuclear cell infiltration adjacent to irregular bone trabeculae, indicating low grade inflammatory reaction.

cent with 3.63 gm. albumin and 4.64 gm. globulin. Serum calcium was 10.2 mg. per cent; phosphorus was 3.72 mg. per cent; alkaline phosphatase was 1.98 Bodansky units. Spinal fluid studies were within normal range and a Kolmer test was nonreactive.

Roentgen findings. Roentgenographic examination on June 7, 1958, showed that, in the interval of eight and one-half years, the first ribs had increased amazingly in density and size (Fig. 4). While the anterior portions of these ribs were especially involved, the entire rib structure on the left and slightly less of the body of the rib on the right were also affected.

In addition, the left fifth, sixth, and seventh ribs were heavily calcified anteriorly. There was considerable distortion of the usually regular trabecular pattern of bone in the involved areas.

Course. Within two weeks the neurologic signs rapidly disappeared and vision improved. The final clinical opinion was that the patient now had multiple sclerosis and a "calcific bone disease of unknown etiology."

Comment. It is felt that the presence of disorders of the skin, endocrine and nervous systems in this case are unrelated to the changes in the thoracic cage. This assumption is based on the absence of generalized bone or joint disease, and the lack of significant abnormality in chemical studies of the blood relating to bone disease. The only persistent abnormality was an elevated globulin in the serum, for which no cause was ascertained. The changes in the ribs are very striking and are similar to descriptions of previous cases of Tietze's syndrome. Cases reported in the literature, however, were followed for much shorter periods of time.

Case II. O.S., a seventy-one year old man with known cirrhosis and diabetes, was admitted to the hospital in June, 1954, complain-

ing of weakness and weight loss of 15 pounds during the preceding five months. He had had transitory pains in the back and legs but, two months prior to admission, he developed moderately severe pain over the left second rib anteriorly. Several weeks after onset of the chest pain, a tender swelling appeared in that area. There was no history of antecedent trauma.

Physical examination. There was a firm protrusion over the left second costochondral junction, which was slightly tender on palpation. Hepatomegaly was also present.

Roentgen findings. Roentgenograms in conventional projections of the chest made on June 9, 1954 showed only a primary calcified complex in the left upper lung. Tangential roentgenograms of the mass overlying the second rib anteriorly revealed a soft tissue tumor projecting from the chest wall (Fig. 6). It contained no calcium and the underlying structures were normal.

Course. The mass over the left second rib increased in size gradually but pain related to this lesion diminished in several weeks. The mass was considered clinically to be an osteochondroma, or callus secondary to undetermined trauma. Because of the increase in size, however, a biopsy was undertaken on August 13, 1954, and, at the time of surgery, the lesion was judged to be of fibrocartilaginous consistency. Microscopic examination showed skeletal muscle infiltrated by mononuclear inflammatory cells, mostly lymphocytes, and bands of connective tissue. This tissue was interpreted to represent a fibrous nodule of the chest wall. A biopsy of the liver revealed hemochromatosis. This was treated by bloodletting. Conventional roentgenograms of the chest made in 1955 revealed no significant abnormalities.

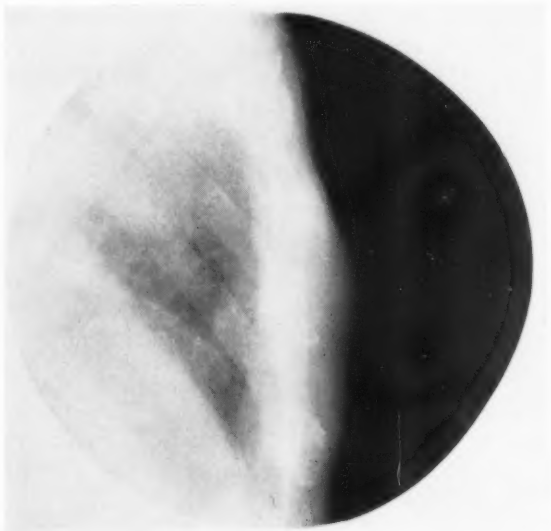

Fig. 6. Case 11. Tangential view shows soft tissue mass on the anterior chest wall, which was not visible on conventional roentgenograms. A biopsy revealed low grade inflammation and fibrosis.

Comment. This case presents a reasonably typical development of Tietze's syndrome, apparently not related to the patient's primary problems of hemochromatosis, with cirrhosis and diabetes.

ETIOLOGIC CONSIDERATIONS

Tietze mentioned that this syndrome might be a form of chondrodystrophy following an upper respiratory infection.[20] Subsequent reports and the cases presented in this paper do not confirm a consistent cause and effect relationship between the skeletal changes and respiratory infections. However, the possibility of trauma incident to coughing, or other sudden but unrecalled stress on the chest wall, has been suggested as a causative factor by several authors.[1,10,11,13,17] Of 65 cases where symptoms of cough or respiratory disease were recorded in Kayser's review,[11] 51 had recent or active upper respiratory infection. All 22 cases reported by Geddes[8] and the 5 published by Gill and his group[9] had either antecedent or coexistent upper respiratory disease. But Düben[6] recorded 10 cases, none of whom had cough or respiratory disease. In a recent report by Karon, Achor, and Janes,[10] only 5 of the 13 cases had cough or respiratory infections associated with the syndrome. Etiologic association of upper respiratory disease and Tietze's syndrome has not been entirely excluded. It may possibly be a viral osteochondritis secondary to infection of the upper respiratory tract.

Malnutrition and specific infections, such as tuberculosis, syphilis, and pyogenic disease have been ruled out as a cause of this complex.[18]

Motulsky and Rohn[16] suggest that microtrauma to the costosternal ligaments is a possible etiologic factor. They refer to the particular anatomy of the ligaments of the second chondrosternal joint, which is the

most frequently involved site in this syndrome. Beck and Berkheiser[1] noted that there appeared to be a buckling forward of the cartilage as it was seen at surgery, and they believe that the process is due to contracture of the ligament lying posterior to the second costal cartilage. Karon and his group[10] feel that unusual local stress on the involved cartilage is an essential feature.

There is a small synovial lined joint at the costosternal junction of the second to the sixth ribs, which tends to be obliterated in middle age.[4] Karon *et al.*[10] remarked that trauma to this joint or to its ligaments may result from a cough and produce the pattern of Tietze's syndrome. However, all of the surgical reports and descriptions of biopsy specimens fail to mention the presence of synovial tissue or other evidence of a joint type of pathology. It is also of interest to note the report by Shanbrom and Feher[19] in which the syndrome is stated to affect a laryngeal cartilage as well as the usual rib site. It is probable that any cartilage may be involved. There is unanimity of opinion in the literature that *no* explanation yet offered accounts adequately for the findings in Tietze's syndrome.

Study of the first case presented in this paper, with particular reference to the roentgen changes, suggests an unusual osteochondritis in which there is perichondral and periosteal elevation with hypertrophy of cartilage and bone. The histologic findings indicate the existence of a low grade inflammatory process. They are similar to those described by a few other authors in cases without gross roentgen abnormalities, but it must be noted that they are at variance with the usually normal microscopic findings reported in the large majority of patients with this syndrome.

ROENTGEN FINDINGS

In the initial report by Tietze, his consultant in roentgenology described enlargement, thickening and increased calcification of the affected ribs in 2 of the 4 cases. One patient was re-examined after a two month interval, and a significant increase in calcification had taken place. However, Tietze questioned whether the calcifications described were a normal physiologic development or were associated with the disease. De Haas[5] mentioned that 1 case of the 8 he encountered had a "densely calcified rib." Leger and Moinereau[14] described an enlargement and porosity of the anterior rib associated with deposition of calcium as seen on roentgenograms of 1 of their 5 cases. Santani and Fujii[18] found, in 1 of their 9 cases, shortening of the rib and calcification of the cartilage. Ordinary calcification, or lack of it, in rib cartilages is not related to the occurrence of the syndrome. Kayser[11] noted in his review that 8 cases out of 21 classified in this respect were stated to have calcified cartilages. An occasional report in the literature of isolated dense ribs may represent burned out areas of Tietze's syndrome involvement.[12] None of the authors have published reproductions of roentgenograms of this condition.

Lindblom[15] stated that all his cases of rib osteochondritis revealed local swellings subcostally, when roentgenograms in tangential projection were made and compared to the normal side. In 2 cases, he noted that there were calcium deposits present, which he could not distinguish from normal cartilage calcification. At operation, 1 of these 2 patients was found to have an abscess, and in the other no pathology was seen in the cartilage or bone. The roentgen features in these cases were not illustrated but the latter may represent an instance of Tietze's syndrome.

In our Case 11, a tangential view of the lesion does not reveal subcostal swelling but demonstrates a soft tissue mass projecting from the anterior chest wall over the costosternal junction (Fig. 6). This would seem to be the most common roentgen change to be anticipated. A great majority of patients with this syndrome who have had chest roentgenograms are reported to show no abnormalities on conventional studies. Since the cartilage is primarily involved, the mass must be quite large to present a noticeable density on the posteroanterior

chest roentgenogram or project far enough to be seen on the lateral roentgenogram. The usual height of the swelling is about 0.5 to 3.0 cm.[7] above the chest wall. This is difficult to detect unless roentgenograms in tangential view are made or calcium deposits are present.

Case 1 illustrates the value of a long term follow-up in that changes described by some authors can then be demonstrated to be transitional to features observed by others. The early changes seen in this case were widening of the anterior ends of the affected ribs with loss of bone pattern and density and with irregular calcium deposition in the broadened cartilages (Fig. 1). The net effect, roentgenographically, seemed to be a shortening of the involved ribs compared to the normal. In time (within four to eight weeks) the process of calcium loss was reversed and increased calcium deposition in the hypertrophied cartilages took place (Fig. 2). This was accompanied by increased bone formation in the more proximal portions of the ribs. An intermediate phase of periosteal elevation with new bone layers was visible on interval examinations (Fig. 3). The subperiosteal new bone and subperichondral cartilage were gradually incorporated into the rib structures. Excessive calcification of the cartilage was then observed as well as hypertrophy of the bony portions of the affected ribs. The final result, in this case, was a striking increase in density and size of most of the involved bones (Fig. 4). In this patient the left clavicle was affected as were several of the ribs.

PATHOLOGY

The gross appearance of the defects has been described as hypertrophy of the cartilage, with or without buckling or forward angulation,[1,9,11,20] or as normal cartilage with thickening of the adjacent soft tissues including the perichondrium.[6,8,20]

Histologic sections are most frequently interpreted as showing normal cartilage. Sometimes there is an alteration of architecture which, however, cannot be considered neoplastic. Several reports indicate the presence of granulomatous or fibrotic change[2,6,14] in the cartilage or nearby tissues. There are isolated contrary reports of vascular insufficiency (Leger and Moinnereau[14]) and of increased vascularity (Gill *et al.*[9]).

The cases presented in this paper showed in one instance the histologic and roentgenologic appearances of inflammatory disease, which can be properly termed an osteochondritis (Fig. 5). The clinical findings and the roentgenologic development of the lesions are quite consistent with the cases of Tietze's syndrome originally described. The second case similarly fits the clinical pattern of the syndrome. The histologic sections showed mononuclear cell infiltration of the muscle over the cartilage and connective tissue bands which formed a thickened nodule with considerable fibrosis. It seems reasonable to conclude that the lesions are inflammatory in origin.

TREATMENT

The disease is self limited and, without treatment, the symptoms in most patients will gradually disappear. Roentgen therapy of unstated quantity and quality was used in 2 cases and was reported to be of little or no benefit.[10] Local procaine injection relieved pain temporarily. Beck and Berkheiser[1] recommended resection of the lesions to achieve relief of symptoms and to provide material for study of the pathology. Celio and Nigst[3] and Karon *et al.*[10] advocate local injection of hydrocortisone (20 to 37.5 mg.). This gave prompt relief from pain and resulted in diminution of swelling in the 7 patients in whom it was tried. This technique is simple, effective and probably the treatment of choice.

SUMMARY

1. Two cases are presented of the syndrome of painful hypertrophy of one or more costal cartilages and bone (Tietze's syndrome) which is a benign self limited condition of unknown etiology. It must be

distinguished from more serious causes of chest pain.

2. It is emphasized that there may be typical and diagnostic alterations of cartilages and ribs. These consist of hypertrophy of the costal cartilages, best demonstrated by roentgenograms in tangential views, and excess calcification of these cartilages. There may also be periosteal reaction with increased size and density of the affected ribs. The second ribs are the most commonly affected.

3. The complex seems to be an unusual osteochondritis of undetermined cause.

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SKELETAL FEATURES VISIBLE ON THE ROENTGENOGRAM OF THE HAND AND WRIST WHICH CAN BE USED FOR ESTABLISHING INDIVIDUAL IDENTIFICATION*

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THE difficulties experienced in Korea and elsewhere in attempting to identify the interred remains of servicemen which were not accompanied by identification tags and which had no distinguishing dental or other features emphasized the need for some additional means of determining the identity of human skeletal remains. On the basis of a rather extensive experience in working with roentgenograms of the hands and wrists of children and adults, the writer had gained the impression that the various skeletal features visible on such roentgenograms might be found to differ sufficiently from one person to another to permit individual identification from them alone. This report summarizes the results of an attempt to determine the usefulness of those skeletal features for this purpose.

It was established that, as seen on the usual posteroanterior roentgenogram, the various bones of the hand and wrist show individual differences in form and in other features which, in the aggregate, are sufficient to distinguish the hand roentgenogram of one person from that of another. Beginning with the ends of the radius and ulna and proceeding distally, some of these features are: (1) the shape and relative size of the styloid processes of the radius and ulna; (2) the shape of the individual carpal bones; (3) the size and shape of the individual metacarpals, the relative width of their cortices and their medullary cavities, the individual differences in the outline of the margin of the cortex which is immediately adjacent to the medullary cavity and the shape and position of the white lines visible in the heads of the metacarpals; (4) the differences in the shape and proportions of the individual phalanges; and (5) the fine details of the trabecular pattern visible in the shafts of the various bones, especially in the proximal and middle phalanges.

ILLUSTRATIVE ROENTGEN FINDINGS

The hand roentgenograms reproduced in Figure 1, *A–H* illustrate some of the individual differences which occur in the distal ends of the radius and ulna, in the individual wrist or carpal bones, and, to some extent, in the proximal ends of the metacarpals, with which the distal row of carpals articulates.

The distal ends of the radius and ulna vary from individual to individual in the relative size and shape of their styloid processes, in the degree of curvature of their opposing borders, and in the shape and relative size of that part of their mutual articular surfaces which is visible on the roentgenogram. These features are shown most clearly in Figure 1, *A*, *B* and *C*, though they can be seen also in the other illustrations. It is apparent from the part of the shaft of the radius which is included in Figure 1*B* that that bone widens more abruptly toward its distal end than do the other radii shown in Figure 1. Similar differences occur in the shape of the shaft of the ulna, and both can be useful features in individual identification.

The carpals often have distinctly different shapes in different individuals. The following are some of the more striking of those differences.

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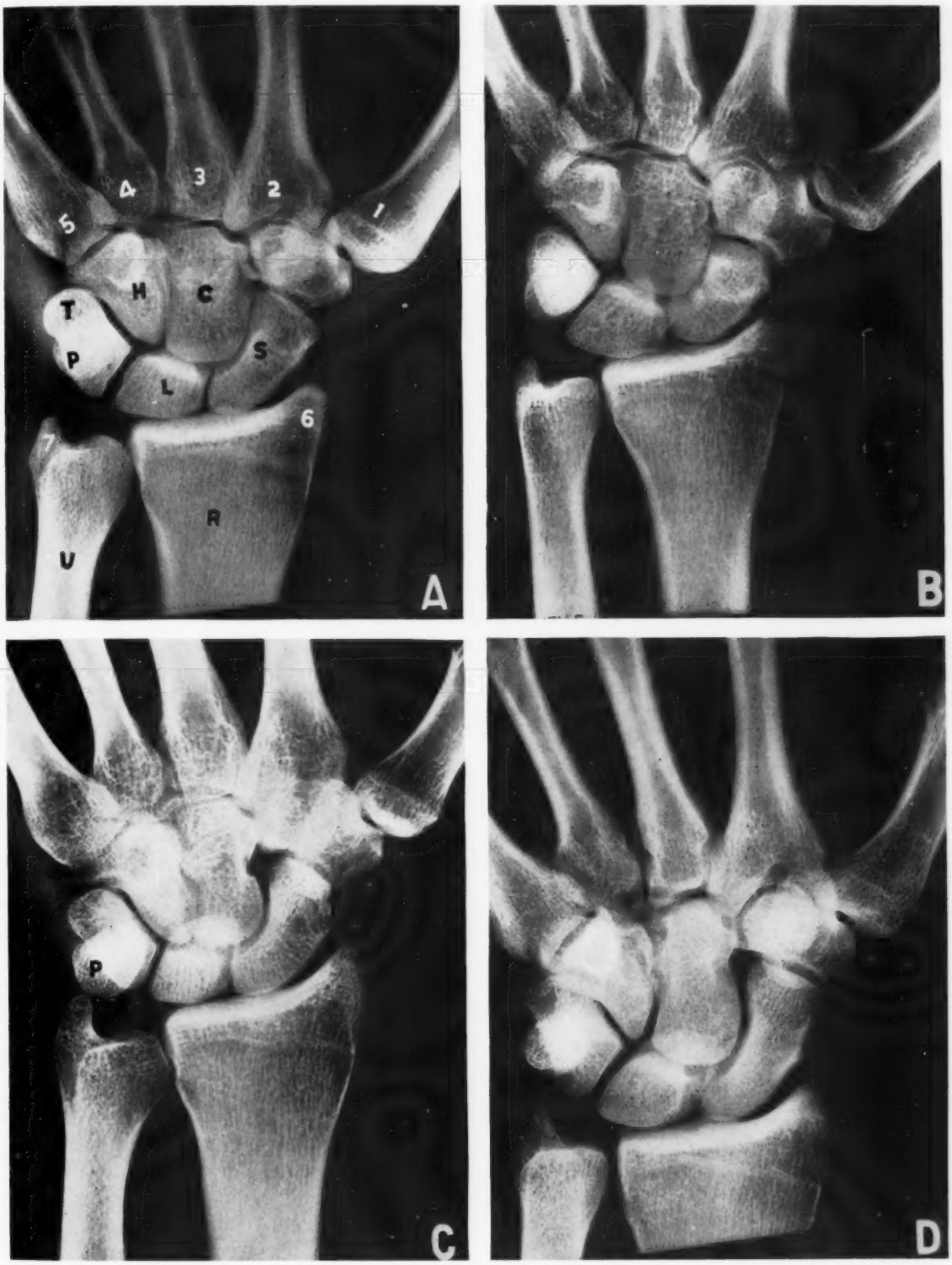

Fig. 1. (*A–D*) 1, 2, 3, 4 and 5—first, second, third, fourth and fifth metacarpals, respectively; 6—styloid process of the radius; 7—styloid process of the ulna; C—capitate; H—hamate; T—triquetral; P—pisiform; L—lunate; S—scaphoid; R—radius; U—ulna.

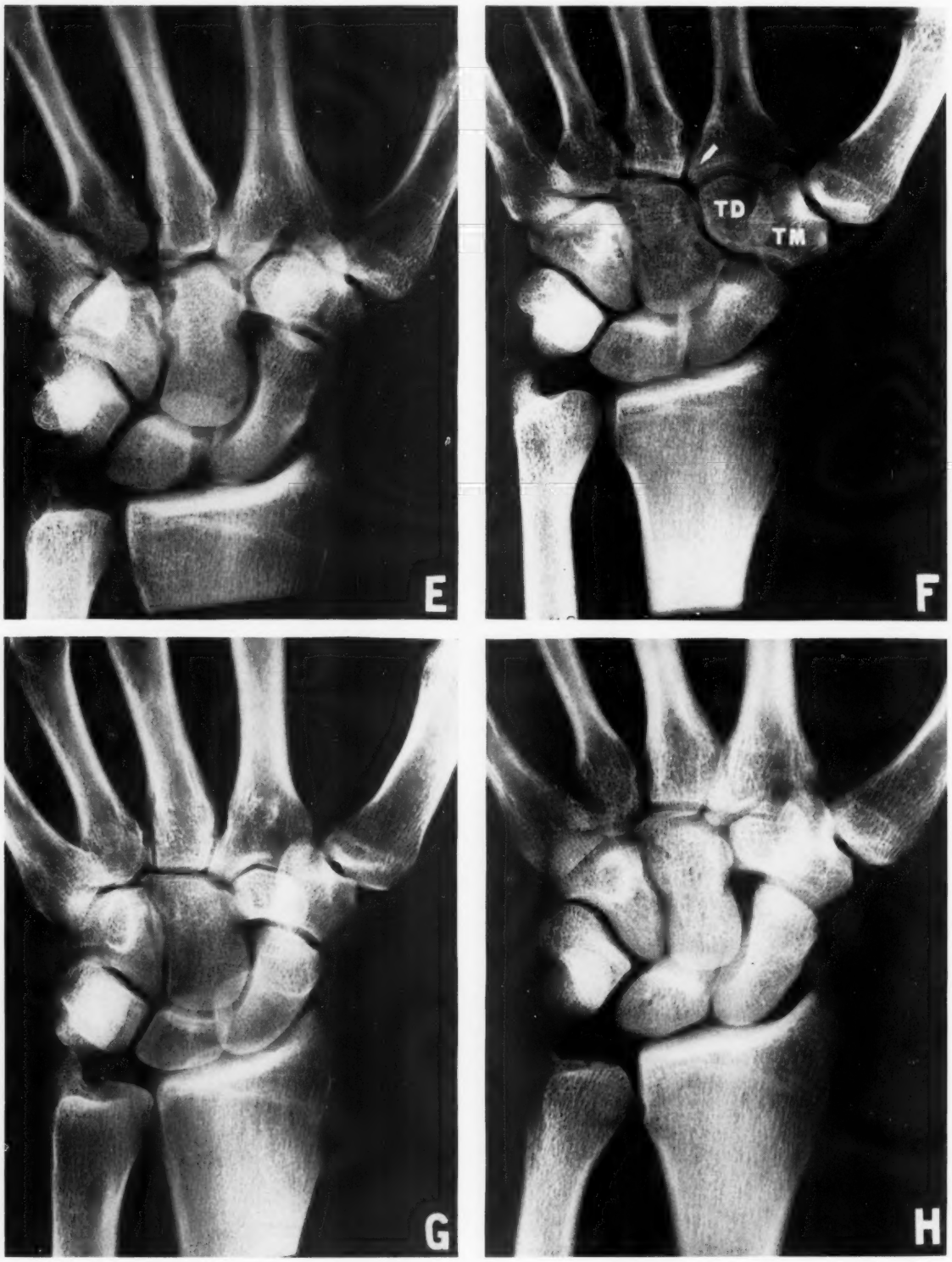

Fig. 1. (*E–H*) TD—trapezoid (lesser multangular); TM—trapezium (greater multangular). (The arrow on the base of the second metacarpal in *F* points to its capitate articular process.)

The capitate may be somewhat elongated, as shown in Figure 1, *D*, *E* and *H*, or it may have a more block-like form like those in Figure 1, *A* and *B*. There may be a well defined notch on the surface adjacent to the scaphoid (Fig. 1, *C* and *D*) or this notch may be absent or at least not visible in the usual projection. Occasionally, as in Figure 1*F*, the proximal part may be widened transversely toward the lateral (radial) side of the hand.

The hamate varies in its proportions (Fig. 1, *G* and *H*), in the degree of constriction of its proximate half (Fig. 1, *A* and *H*) and in the roundness or angularity of its proximal tip (Fig. 1, *C*, *D* and *E*). There are differences, too, in the shape and position of the shadow of its hook or hamulus, which can be seen in the distal half of the bone as a triangular (Fig. 1 *D*) or oval (Fig. 1 *G*) area bounded by a thick, white line. Figure 1, *B*, *C*, *G* and *H* shows contrasting types of hamates.

The outline of the triquetral is often partly obscured by the pisiform, the shadow of which intrudes to a varying extent upon it, and to which it has a variable size relationship. Marked differences occur, also, in the shape of its distal (Fig. 1, *A* and *F*), its medial (Fig. 1, *B* and *E*), and its lunate borders (Fig. 1, *C* and *D*), and in the shape of its proximal tip (Fig. 1, *G* and *H*).

In the lunate, variations frequently occur in the shape of its proximal and triquetral borders and in the extent to which its proximo-medial tip extends toward the latter bone. Examples of such variations can be seen in Figure 1, *A*, *B*, *D*, *G* and *H*.

The scaphoid (navicular) may be relatively narrow and elongated (Fig. 1, *D* and *E*), or shorter and broader (Fig. 1, *B* and *F*). The slight projection on its lateral border, which marks the approximate distal limit of its radial articular surface, varies in distinctness and in its position with reference to its distal end (Fig. 1, *B*, *D* and *F*).

On the posteroanterior hand roentgenogram the trapezium (greater multangular) is often partly obscured by the trapezoid (lesser multangular), by the distal end of the scaphoid, and by the bases of the first and second metacarpals. There are detectable differences, too, in the shape of the margin of that portion of the bone which overlies the lateral (radial) side of the base of the second metacarpal and also in the shape of its free, lateral margin. In Figure 1 *G* the distal tip is definitely less rounded than it is in Figure 1, *A*, *D* or *E*. The proximal tip of the trapezium in Figure 1 *F*, which on that roentgenogram just touches the distal margin of the scaphoid, is smaller and much less rounded than the corresponding structure in Figure 1, *D* or *E*. Differences in the shape of the lateral margin of this bone are evident in Figure 1, *C*, *F* and *H*.

The trapezoid (lesser multangular) can vary greatly in its relative proportions. Contrast the almost circular trapezoid in Figure 1 *B* with the squat, transversely elongated one in Figure 1 *G*. Its distal margin conforms to the variable shape of the surface of the base of the second metacarpal, with which it articulates.

Closely associated with the trapezoid is the process of the base of the second metacarpal which extends proximally to articulate with the corresponding facet of the capitate. Some idea of the variety of shapes which this process can take can be obtained by comparing *A*, *B*, *E*, *F* and *G* in Figure 1.

On the posteroanterior roentgenogram of the hand, the second, third, fourth, and fifth metacarpals are seen, whereas the first metacarpal appears in the oblique view. In such roentgenograms, the individual metacarpals vary somewhat in form from one person to another, as is shown in Figure 2, *A–F*. In Figure 2 *F*, the middle part of the shaft is slightly constricted and the ends are relatively wide. In Figure 2, *C* and *D* the sides are straighter and lack any marked central constriction. Since, as mentioned above, the shape and relative size of that portion of the base of the second metacarpal which articulates with the capitate vary considerably in different individuals, those features, too, are

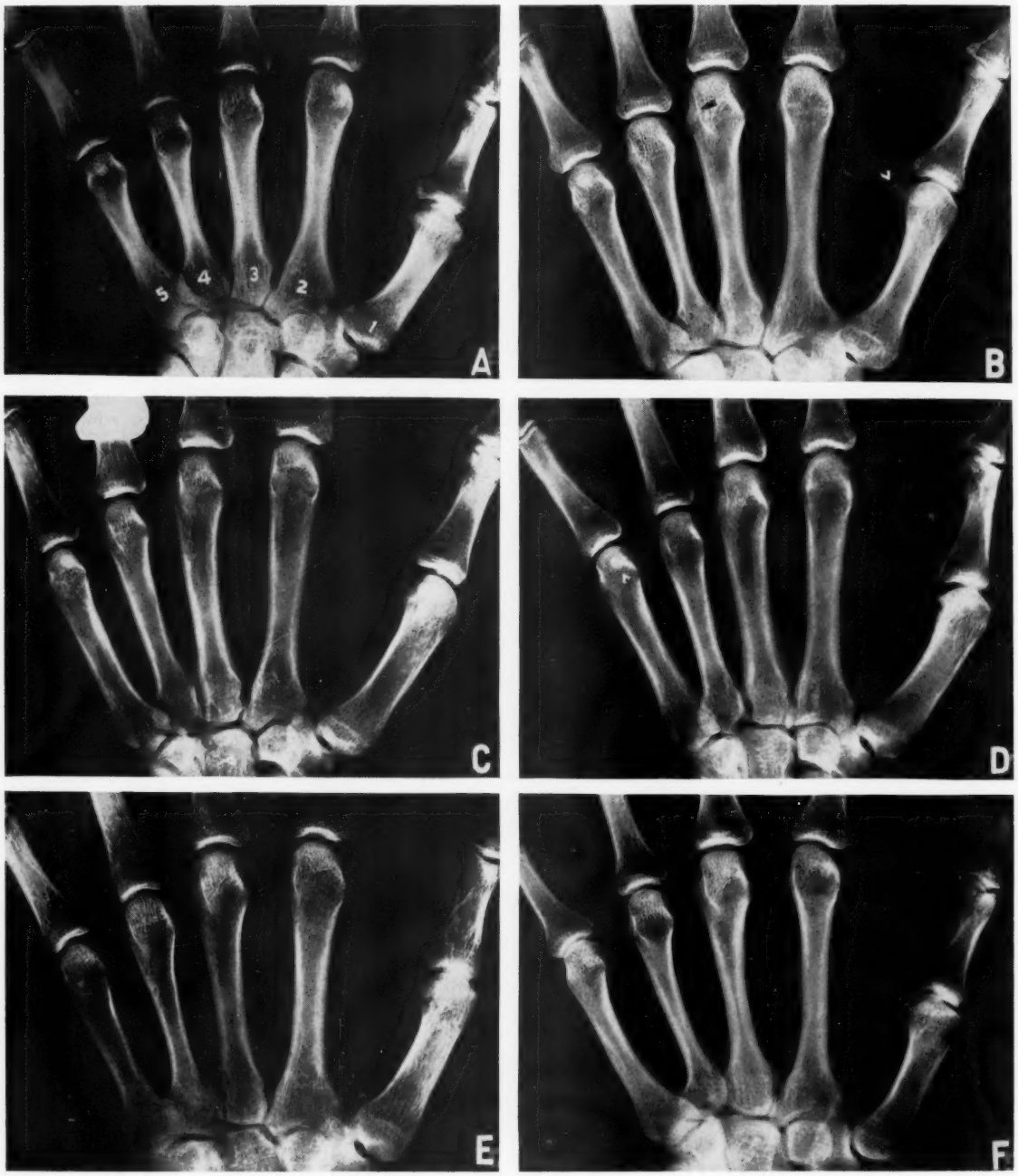

Fig. 2. (*A–F*) 1, 2, 3, 4 and 5—first, second, third, fourth and fifth metacarpals, respectively. The black pointer on the head of the third metacarpal in *B* indicates one of the white lines referred to in the text. The white "v" just medial to the head of the first metacarpal points to the faint outline of one of the two sesamoid bones which regularly occurs there. Their shape and relative size are occasionally helpful in identification. The inverted "v" on the head of the fifth metacarpal in *D* points to one of the metacarpal sesamoid bones which vary in number as well as in relative size in the hands of different individuals.

often very helpful in establishing identification.

In addition to differences in their shape, there are other—often striking—differences in the metacarpals, as seen on the hand roentgenogram. One of the most useful of these for purposes of identification is the relative width of the cortex—the com-

pact bone which makes up the wall of its shaft—and of the medullary cavity which it encloses. Occasionally, as in Figure 2 *F* (especially in the third metacarpal), the cortex is relatively thick and the size of the medullary cavity in the central portion of the shaft appears to be considerably reduced. Figure 2 *C* shows metacarpals in which this relationship is quite different, due to their relatively thinner cortex and the lack of constriction of the central portion of their shafts. In such metacarpals, the medullary cavity is relatively wide and the cortex is correspondingly thinner. As can be seen in the illustrations, the relative width of the cortex and of the medullary cavity can differ somewhat from one metacarpal to the other in the same hand.

An individualizing feature of the hand roentgenogram to which, it is believed, attention has not previously been directed is the shape and thickness of the white lines located on or near the medial and lateral borders of the heads of the metacarpals. In the third metacarpal in Figure 2 *B*, this feature is visible as a fine white line which begins near the distal, medial margin of the head and extends proximally in a gentle curve and ends near the midline. The corresponding structure on the lateral (thumb) side of the same metacarpal head is a thicker and shorter white linear mark, the proximal end of which appears to be continuous with the lateral margin of the cortex. These marks have a somewhat different configuration and extent in the other metacarpals of the same hand and in the corresponding metacarpals of the other hand shown in Figure 2. In Figure 2 *D*, for example, the mark on the ulnar side of the head of the third metacarpal takes the form of an even finer, slightly concave line, the concavity of which is directed medially. In the third metacarpal in Figure 2*F*, the proximal part of the corresponding line makes an obtuse angle with its more distal portion. Similar lines are visible on the heads of the other three metacarpals and, if studied carefully, minor, but distinct, differences between them will be noted.

These lines develop separately, though concomitantly, in the epiphysis and in the adjacent part of the metacarpal shaft, during childhood, and their two parts become continuous only when epiphysial fusion is completed. Once established, they seem to remain practically unchanged throughout the life of the individual. Even minor differences in their shape and extent can be of considerable help in identification.

A careful examination of stereoscopic roentgenograms of dried metacarpals disclosed that these white lines are composite shadows to the formation of which structures on the volar and dorsal surfaces and others which are located between those surfaces make varying relative contributions in different bones.

The shape of the base of the fifth metacarpal is subject to considerable variation. Sometimes, its medial border is rather bulbous, as in Figure 2 *D*, flaring, as in Figure 2 *E*, or relatively straight, as in Figure 2 *A*. The shape of the base of the third and fourth metacarpals is often obscured by the shadows of adjacent structures but when, as in the third metacarpal in Figure 2, *A* and *B*, their proximal margins are visible, they, too, show individual differences in form. The width of the proximal margin of the base of the third metacarpal appears to be related to the size and shape of the proximally projecting capitate articular process of the second metacarpal, to which reference has been previously made. When the latter is relatively wide and directed somewhat medially, as in Figure 2 *A* this part of the base of the third metacarpal tends to be rather narrow, and its lateral margin conforms to the shape of the intruding process.

The individual phalanges, especially the proximal and the middle ones, also possess roentgenographic features which serve to individualize them. These include not only differences in external form but also finer differences in their trabecular pattern. Some of these features can be seen in Figure 3, *A–H* which provides a somewhat enlarged view of the basal phalanx of the

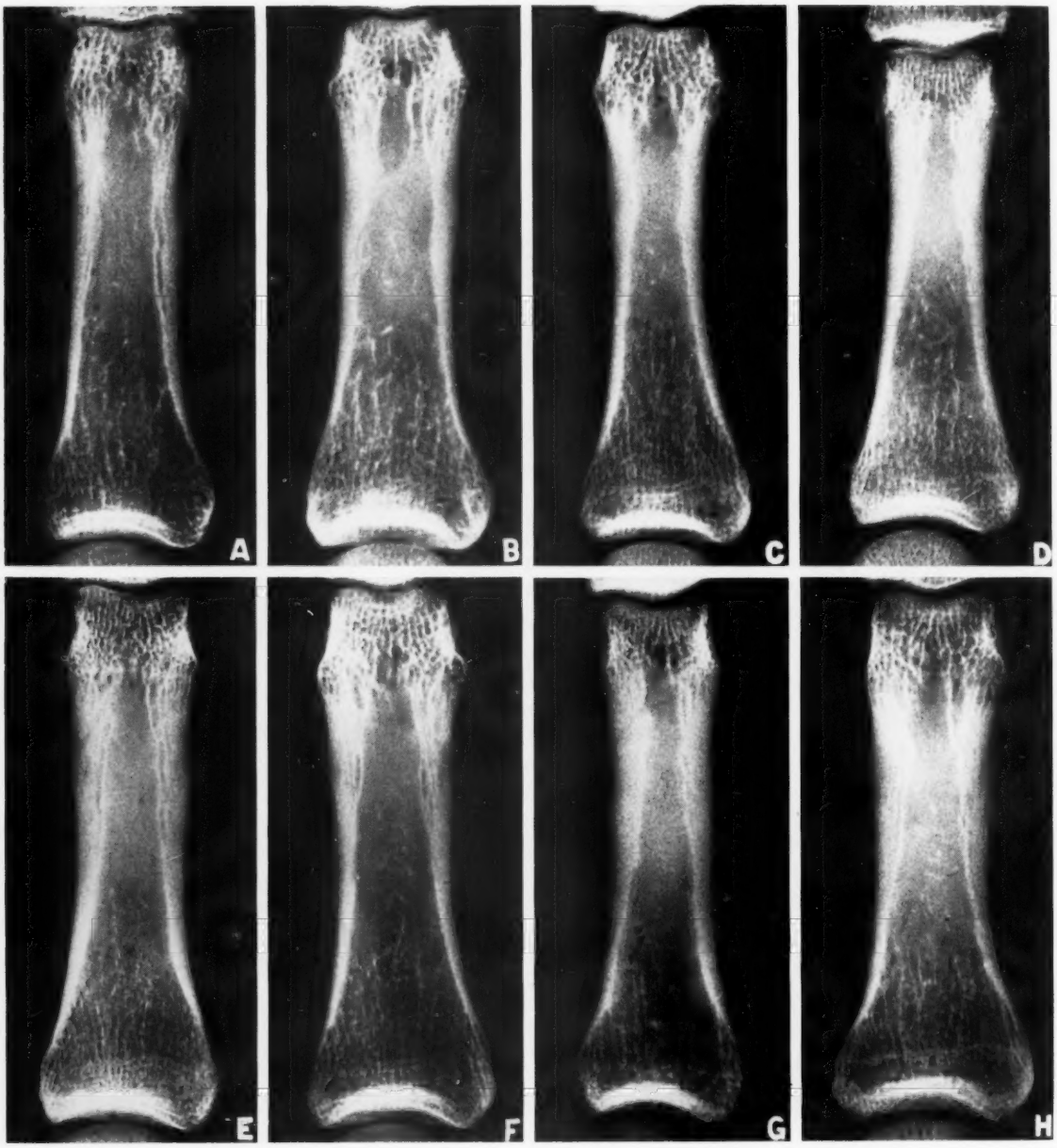

Fig. 3. (*A–H*) Roentgenograms of the basal phalanx of the third digit of 8 young men showing individual differences in the form and in the details of their internal structure (2×).

third digit of the left hand of eight different young men. In some, the middle of the shaft is slightly constricted, as seen in Figure 3 *C*, while in others the sides are straighter, as shown in Figure 3 *B*. The phalanges differ, too, in the absolute and relative size of their cortex and medullary cavity. The cortex appears on the roentgenograms as the dense white material which composes the borders of the phalanges and extends for a varying distance centrally (Fig. 3, *A*, *C* and *G*).

The innermost part of the cortex, *i.e.*, the portion immediately adjacent to the central cavity, can vary markedly in form from one individual to another. In some, as in Figure 3 *C*, there is no apparent separation between the innermost layer of the cortex and that part which lies external to it. In others, as in Figure 3, *A* and *E*, the

innermost layer of the cortex is separated from that peripheral to it by what appear to be irregular, elongated spaces that become more open as one follows them toward the distal end of the phalanx—the part which is toward the top of the figure. In addition to these features, there are often striking differences in the size, shape, and arrangement of the fine trabeculae which appear on the roentgenograms as irregular, white linear markings, the long axis of most of which tends to parallel that of the phalanx. They are rather irregularly distributed throughout the length and breadth of the medullary region but tend to be more numerous or more conspicuous toward the ends of the phalanx.

The coarser of these trabecular markings remain practically unchanged from early adolescence, when their pattern first becomes stabilized, until well past middle life. In some individuals, however, the pattern can be obscured or modified by bone changes associated with aging. Unless these changes are quite extensive—and in many old people they are not—enough of the original trabecular pattern usually remains so that it can be compared satisfactorily with that visible on a roentgenogram which had been made of the same hand before the degenerative changes occurred.

DISCUSSION

Though, for purposes of illustration, various features of only the basal phalanx of the third digit have been considered here, the other basal phalanges and the middle phalanges as well show similar individual patterns. Occasionally, there are also comparable features of the distal phalanges which are helpful in identification. It should, of course, be remembered that identity is not usually established from the features of a single bone, but only by comparing carefully the form of all the individual bones that are visible on the hand roentgenogram.

In addition to the structural details which we have been considering, there are differences, too, in the total impression that one obtains from viewing the entire hand roentgenogram. This impression derives, in part, from individual differences in the size and what might be called the degree of robustness or gracility of the bones of the hand and wrist, which alone is often sufficient to enable one quite readily to distinguish one hand roentgenogram from others with which it is being compared.

On a roentgenogram of the hand and wrist, 27 complete bones and parts of two others (the distal ends of the radius and ulna) are visible. Since, as has been shown in this paper, most of them possess a number of structural features which can differ from one person to another, the chance of the hand roentgenograms of any 2 persons being identical in all of those features would seem to be very small—if, indeed, such identity ever occurs, even among identical twins.

Professor Harold E. Jones, Director of the Institute of Child Welfare of the University of California at Berkeley, kindly permitted the writer to study the roentgenograms of the right and left hands of 70 pairs of like-sexed twins on whom he had previously made some other observations. Approximately 40 of those pairs of twins appeared to be identical.

While there was a very striking resemblance between the hand roentgenograms of the 2 members of each presumably identical pair, there were, in every instance, some features which made it possible to distinguish the hand and wrist bones of one person from those of his or her twin. In some cases, however, the over-all similarity between them was very great, indeed. For this reason, and because the number of identical twins whose roentgenograms were studied is so small, the possibility remains that skeletal features which are adequate to distinguish between the hand roentgenograms of unrelated persons and of ordinary siblings might, in some instances, not be sufficiently discriminating to distinguish the hand roentgenograms of one identical twin from those of the other member of the

pair. However, as mentioned above, no such instance was encountered among the cases available to the writer.

In approximately 500 young men, whom we recently examined roentgenographically, roentgenograms were made of both the right and left hand. Though, as might be expected, an individual bone of one hand or wrist was not always identical in form with the corresponding bone of the other side, it was found that there is a sufficiently close over-all similarity in the shape and proportions of the bones of the two hands to permit the two roentgenograms of the same man to be paired successfully, when all identifying marks are covered and the roentgenograms are examined in a random order. It should, therefore, be possible to identify skeletal remains in which the bones of the forearm, wrist, and hand of only one side are present by comparing their roentgenographic features with those visible on a hand roentgenogram which had previously been made of the same or of the opposite side.

It might be thought that the ease with which the hand roentgenogram of one person can be distinguished from that of another in our population is, to some extent, due to the heterogeneity in race and in national antecedents of the people of the United States. Our study of the hand roentgenograms of several hundred Apache Indians from the White River Reservation in Arizona and of a larger number of American-born Japanese living in California established, however, that comparable individual differences in the skeletal features which we had observed in the more heterogeneous white population of this country exist also among American Indians and Japanese. The hand roentgenogram can, therefore, be used satisfactorily for individual identification within those groups and, presumably, within other racial groups as well.

CONCLUSION

These findings demonstrate that it is quite possible to establish the identity of an individual from the skeletal features visible on a roentgenogram of his hand and wrist. Such roentgenograms, therefore, can provide a valuable supplement to the photographic, dermatoglyphic, and other records which are ordinarily used for identification. Since many of the skeletal features visible on the hand roentgenogram remain recognizable in the skeleton even after years of burial, the hand roentgenogram can also provide conclusive proof of the identity of persons whose remains lack all other identifiable features.

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Forty-second Annual Meeting: The Caribe Hilton Hotel, San Juan, Puerto Rico, March 17–19, 1960.

EDITORIAL

THE INTERNATIONAL CONGRESS OF RADIOLOGY AND THE INTERNATIONAL SOCIETY OF RADIOLOGY

ORGANIZATION

THE International Congress of Radiology is organized according to provisions set forth in the "Rules and Regulations" which function as a Constitution and By-Laws. This document establishes three important Committees by which the Congresses are governed, namely: (1) the National Board of Management, (2) the International Committee, and (3) the International Executive Committee.

The National Board of Management of the host country has complete responsibility for the organization, management and financing of any Congress. It is composed of the President, chosen by the delegation of the host country at the time its invitation is accepted; of the Secretary General and other officials appointed by the President and has a headquarters office in the City in which the Congress is to be held.

The International Committee is vested with the ultimate authority of the International Congress. It is composed of the Delegations appointed by the National Radiological Society or Societies of all countries which desire to participate in the Congress. Each delegation consists of not more than five individuals, of whom one is selected as Chairman. Each delegation has one vote, voiced by its Chairman. The meetings of the International Committee are held prior to the opening of the Congress, subject to the call of the President.

Because of the large size of the International Committee, an International Executive Committee of eleven individuals was established. The Executive Committee is composed of the President, of the last three living past Presidents and of the Chairmen of the National Delegations of seven countries selected by mail vote by all National Delegations registered at the Headquarters office of the Congress. Prior to the International Committee meeting, the Executive Committee discusses international problems affecting the Congress and prepares recommendations for the larger body. It acts much as a steering committee.

HISTORY OF THE CONGRESSES

The First International Congress of Radiology was organized in London in July, 1925. It was intended to provide an opportunity for radiologists of various countries to become personally acquainted with each other and to discuss scientific problems common to Radiology in all nations. The President was Mr. C. Thurstan Holland, an eminent British Radiologist.

At the London meeting, nine resolutions were adopted by the International Committee which established the basic organization of the Congress and which were later formally arranged as the "Rules and Regulations" of the Congress.

The Second International Congress was held in Stockholm during the summer of 1928, with Professor Gösta Forssell as President.

The Third International Congress was held in Paris in the summer of 1931. Dr. Antoine Béclère was President.

The Fourth Congress took place in Switzerland in the summer of 1934, with Professor Hans Schinz as President. The meeting opened in Zurich and later moved

to St. Moritz, high in the mountains.

The Fifth Congress was held in Chicago in August, 1937. Dr. Arthur C. Christie was President.

The invitation from Germany to hold the Sixth Congress in Berlin in 1940, under the Presidency of Professor Hermann Holthusen, was accepted. The Second World War interfered and many records were lost.

After the war was over, the British Radiologists wisely decided to revive the International Congress. With wholehearted international cooperation the Sixth Congress was held in London in 1950 with Professor Ralston Paterson as President.

The Seventh Congress was held in Copenhagen in 1953 under the Presidency of Professor Flemming Møller.

The Eighth Congress was held in Mexico, D. F. in 1956, with Professor Manuel Madrazo as President.

The Ninth Congress occurred in Munich in 1959 with Professor Boris Rajewsky, Ph.D., as President.

THE INTERNATIONAL COMMISSIONS

One of the problems confronting radiology in the early twenties was the establishment of a unit of measurement of x-rays. Prior to the First Congress this problem was studied independently in several countries. At this time a Committee for the standardization of x-ray measurements in the United States was organized with Dr. Edwin C. Ernst as Chairman. A year or more before the First Congress, this Committee met in Washington. Without difficulty the co-operation of Mr. Herbert Hoover, then Secretary of Commerce, and of the Director of the National Bureau of Standards, which was under Mr. Hoover's jurisdiction, was secured. The interest of the Director of the Budget was more difficult to arouse. However, with the help of Mr. Hoover an appropriation of thirty thousand dollars was obtained from Congress for the purpose of establishing a section in the Bureau of Standards in which x-ray measurements and the necessary instruments could be standardized.

Similar Committees in other countries, notably in France and in Germany, were working on the same problem and produced units of measurements which were different from each other. At the time of the First International Congress, one of the problems of immediate importance was the establishment of a unit of measurement of x-rays which could be understood and reproduced in all countries. During this First Congress a combined meeting of the Sections on Physics and on Therapeutic Radiology was held to discuss the establishment of an International Unit of Measurement.

At the suggestion of Antoine Béclère, of Paris, a Committee was appointed to nominate qualified physicists and radiologists from various countries for membership on an International Committee to which was assigned the problem of developing a unit of measurement suitable for use throughout the world. Accordingly, Thurstan Holland appointed "The International X-Ray Units Committee" with instructions to report at the next Congress.[1]

The International X-Ray Units Committee made its first report in 1928 at the Second Congress in Stockholm, and established the "roentgen" or the "r" as the "unit of x-radiation" which could be reproduced in all countries. It was promptly adopted internationally and with minor modifications is still in use. This Committee later became known as the International Commission on Radiological Units (ICRU), which in 1953 established the "rad" as the unit of absorbed dose of radiation.

The International X-Ray and Radium Protection Committee was organized at the Second Congress in Stockholm under essentially the same arrangements which prevailed when the Units Committee was appointed in London three years earlier. This Committee later became the International Commission on Radiological Pro-

[1] Taylor, Lauriston S. History of the International Commission on Radiological Units and Measurements (ICRU), Health Physics, 1958, *1*, 306–314.

tection (ICRP).[2] The latest report of this Commission was adopted September 9, 1958.[3]

It is important to emphasize that the members of both of these Committees are selected and appointed on the basis of their individual qualifications for the work to be done, without political considerations.

Because of the difficulty of comparing the results of radiation therapy of malignant diseases in different countries, an International Commission on the Staging of Cancer (ICSC) was appointed in 1950. Its purpose was to establish criteria for the classification of malignant diseases which could be used in reporting the results of treatment. Thus the therapeutic results of one writer might be comparable with those of another. The Chairman was Professor James Heymann of Stockholm. Following his death, Professor Hans Schinz of Zurich became Chairman.

At the Ninth Congress in Munich, an International Commission on Rules and Regulations (ICRR) was established, the purpose of which is to revise the basic regulations which control the activities of the Congresses, with Ross Golden as Chairman.

INTERNATIONAL SOCIETY OF RADIOLOGY

The International Commissions had been meeting immediately before and during each Congress. These Commissions had so much to do that extremely long, fatiguing sessions had to be held. This led Professor Heymann to suggest to the International Executive Committee in 1953 that arrangements should be made for the International Commissions to meet during the three year interval between Congresses when the Chairmen found such meetings necessary. Interval meetings would require financial support. As a mechanism for supporting these Commissions, an International Society of Radiology was proposed which should have a membership of National Radiological Societies rather than of individuals and should establish a headquarters office. The dues paid by member-societies should be used only for the support of these International Commissions. The work of the Commission on Protection as well as on Units had become of increasingly great importance following the development of artificial radioactivity. Efforts had been made by some of the pseudo-governmental international medical and scientific organizations to get control of these Commissions which were originally established by the International Congress through the cooperation of radiologists from many countries. Had this happened, control of these Commissions and of the appointment of their members would have been lost to international radiology.

For these reasons, the International Committee at the Seventh Congress in Copenhagen approved the International Society of Radiology (ISR) in principle and appointed an International Organizing Committee to prepare "Statutes" under which the new organization could operate.

At the Eighth Congress in Mexico, the International Committee adopted the "Statutes" prepared by the International Organizing Committee and the ISR began to function. Its President is the President of the Congress. Its ultimate authority is the "Assembly" which is composed of Chairmen of the Delegations of all countries which have dues-paying member-societies. The "Council" is made up of members of the International Executive Committee and acts as a steering committee. In addition, a "Working Committee" of three members of the Council is at the disposal of the President for the purpose of making decisions between regular meetings of the Assembly which are three years apart.

Professor Flemming Nørgaard, Secretary General of the Seventh Congress, was appointed Secretary-Treasurer of the ISR. His office, in Copenhagen, Kommunehospitalet, Denmark, became the headquarters office.

[2] Taylor, Lauriston S. History of the International Commission on Radiological Protection (ICRP), Health Physics, 1958, *1*, 97–104.

[3] Recommendations of the International Commission on Radiological Protection, New York, Pergamon Press, 1959.

Following the second World War, the *Council for International Organizations of the Medical Sciences* (CIOMS) was established with headquarters in Paris. The function of the Council is to correlate and to assist International Medical meetings. As of 1950, the International Congress of Radiology was represented on the CIOMS by Ralston Paterson, President of the Sixth Congress. At present Henri Maisin, a Belgian therapeutic radiologist, is Chairman of its Executive Committee and Ralston Paterson is a member. International Radiology is, therefore, well represented. World Health Organization and United Nations Educational Scientific and Cultural Organization, as well as other International Organizations, are represented on the CIOMS.

In 1957 Professor Flemming Nørgaard, Secretary-Treasurer of the ISR, was requested to describe the structure and purposes of the ISR to the CIOMS. The ISR was requested to replace the International Congress of Radiology as a member of the CIOMS. This Council has, in the past, made financial contributions to the Congress, in particular to support certain special conferences. In previous years, the Congresses have made small contributions to the CIOMS. Now that the ISR is the representative of world radiology, it will be expected to contribute subscriptions of three hundred dollars per year to the CIOMS which is actually considerably less than the contributions of the CIOMS to the Congress. Representation on the CIOMS is of great importance to International Radiology.

At the time this is written, the ICR had member-societies in thirty-four countries. The dues are twenty-five cents United States money, or its equivalent in any national currency, per dues-paying member of each member-society. Sufficient money had been received by the summer of 1958 to permit the payment of five hundred dollars to each of the three Commissions, the ICRU, the ICRP and the ICSC. Obviously, this is only token support for these important Commissions from International Radiology. However, this support has proved to be of great importance. These Commissions must obtain much larger sums of money from certain Foundations and elsewhere.

Lauriston S. Taylor, Ph.D., Chairman of the ICRU and member of the ICRP, explained to the Board of Chancellors of the American College of Radiology that the International Congress, which meets in a different place once every three years without real continuity, was not regarded as stable backing. Dr. Taylor (1958) states ". . . the ICRP and the ICRU were established, and thereafter sponsored, by the International Congresses of Radiology. In a sense the two Commissions were the only continuing bodies of the Congresses since technically the latter existed only every three years. To some degree this was awkward for the Commissions in that their sponsorship appeared to be uncertain. This situation was alleviated by the organization of the International Society of Radiology in 1956. This society will in the future sponsor . . . the ICRU and the ICRP. By its own international character its sponsorship will carry more weight. As a further outgrowth of the new organization, it has made funds available to the Commissions to assist in their work." Dr. Taylor added that, although small, these contributions proved to be helpful in the approach to certain organizations for larger sums of money. The international importance of these Commissions is emphasized by the fact that they are consulted by the World Health Organization, by the United Nations and other International organizations. Although it is still young, the International Society of Radiology, a continuing organization with a headquarters office in constant operation, has already demonstrated its value in assisting these International Commissions and in keeping them under the aegis of Radiology.

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SOCIETY PROCEEDINGS AND NEWS ITEMS

GOLD MEDALS AWARDED BY THE AMERICAN COLLEGE OF RADIOLOGY

At the Convocation ceremonies of the Annual Meeting of the American College of Radiology held at the Roosevelt Hotel in New Orleans, Louisiana on February 5, 1960, two Gold Medals, the highest award of the College, were presented by Dr. Earl E. Barth of Chicago, Illinois, Chairman of the Board of Chancellors.

Dr. L. Henry Garland, radiologist from San Francisco, California, and Dr. Lawrence Reynolds, radiologist from Detroit, Michigan, and Editor of this JOURNAL, were the recipients of this award for many outstanding contributions to the College "and the profession for which it stands."

OFFICERS OF THE AMERICAN COLLEGE OF RADIOLOGY

At the Annual Meeting of the American College of Radiology held at the Roosevelt Hotel in New Orleans, Louisiana, on February 5, 1960, the following officers were elected: President, Dr. Earl E. Barth, Chicago, Illinois; Vice President, Dr. Robert C. Pendergrass, Americus, Georgia; Secretary-Treasurer, Dr. Fay H. Squire, Chicago, Illinois (re-elected); Chairman, Board of Chancellors, Dr. Arthur J. Present, Tucson, Arizona.

The Executive Director of the American College of Radiology is William C. Stronach, 20 North Wacker Drive, Chicago 6, Illinois, and the Director of Public Relations is Hugh M. Jones, Chicago, Illinois.

The new Chancellors elected are: Dr. Stanley M. Wyman, Boston, Massachusetts (replacing Dr. Earl E. Barth); Dr. Wendell G. Scott, St. Louis, Missouri (replacing Dr. Howard B. Hunt, Omaha, Nebraska); Dr. Alan Sampson, Philadelphia, Pennsylvania (from the Council);

Dr. L. Henry Garland, *left*, is presented the Gold Medal of the College by Dr. Earl E. Barth, *right*.

Dr. Lawrence Reynolds, *left*, receives congratulations from Dr. Earl E. Barth, *right*, on his award of the College's Gold Medal and, in turn, congratulates Dr. Barth as the newly elected president of the American College of Radiology.

Dr. David S. Carroll, Memphis, Tennessee (from the Radiological Society of North America); and Dr. Edward A. Petrie, St. John, New Brunswick (from the Canadian Association of Radiologists).

EASTERN RADIOLOGICAL CONFERENCE

The Eastern Radiological Conference, sponsored by The New England Roentgen Ray Society, will be held on April 21, 22, and 23, 1960 at the Sheraton Plaza Hotel, Copley Square, Boston, Massachusetts.

The program will feature several symposia from the Harvard Medical School, The Peter Bent Brigham Hospital, The Massachusetts General Hospital and Massachusetts Institute of Technology. Selected individual papers will also be included. Tours to the MIT Nuclear Reactor, the 6 billion volt Cambridge Accelerator, High Voltage Engineering Company and the AVCO Research and Advanced Development plant are being arranged. The scientific program is on Friday and concludes Saturday at noon.

The registration fee is $30.00 which includes the annual banquet, reception and tours; twenty dollars is the fee for wives who participate in the ladies' program.

Radiologists and others interested may obtain further information by writing to: Dr. John E. Gary, Secretary, New England Roentgen Ray Society, 1180 Beacon Street, Brookline, Massachusetts.

SURGEON GENERAL'S OFFICE TO PUBLISH RADIOLOGICAL VOLUME SOON

The Historical Unit of the Surgeon General's office of the United States Army Medical Corps is to publish soon a volume devoted entirely to radiology in the United States Army Medical Corps.

Dr. Kenneth Allen requests that all American radiologists who received a citation or decoration from either the American services or foreign countries and who have not already done so pursuant to the appeal published in this JOURNAL in August, 1958 on page 348 should lay aside their personal modesty and report those honors or decorations to him.

It would be of great help, also, if any radiologist who knows of enlisted personnel in their departments who had received decorations or commendations would report those too. Immediate response would be appreciated to the address: Kenneth D. A. Allen, Editor, History of Radiology in World War II, 800 Washington St., Denver 3, Colorado.

SYMPOSIUM ON RADIOACTIVITY IN MAN AT VANDERBILT UNIVERSITY

The Section on Medical Sciences of the American Association for the Advancement of Science is sponsoring a Symposium on Radioactivity in Man, Measurements and Effects of Internal Gamma Ray Emitting Radioisotopes, to be held at Vanderbilt University on April 18 and 19, 1960.

This conference was made possible by a grant from the United States Steel Foundation to the American Association for the Advancement of Science and is supported in part by the Division of Biology and Medicine of the Atomic Energy Commission, the Public Health Service and the Army Medical Research and Development Command.

A fine program has been arranged. Those interested please write to: Symposium Chairman, George R. Meneely, M.D., School of Medicine, Vanderbilt University, Nashville 5, Tennessee.

35TH CONGRESS OF THE PAN AMERICAN MEDICAL ASSOCIATION

The 35th Congress of the Pan American Medical Association convenes in Mexico City this spring, May 2–11, 1960.

Among the fifty Sections included are the Sections on Diagnostic Roentgenology and Radiation Therapy under the Chairmanship of Dr. Eugene P. Pendergrass and Dr. Juan del Regato. Both are extremely anxious for a highly representative participation in each Section. Please forward all communications to Philip J. Hodes, M.D., Jefferson Medical College Hospital, Department of Radiology, Philadelphia 7, Pennsylvania.

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

The Essentials of Roentgen Interpretation. By Lester W. Paul, M.D., Professor of Radiology and Chairman of the Department of Radiology, The University of Wisconsin Medical School; and John H. Juhl, M.D., Associate Professor of Radiology, The University of Wisconsin Medical School. Cloth. Price, $25.00. Pp. 839, with 1203 illustrations. Paul B. Hoeber, Inc., Medical Book Department of Harper & Brothers, 49 East 33rd Street, New York, N. Y., 1959.

The authors of this volume have designed it to bridge the gap between an elementary text and a multiple volume reference work. They have done this admirably. They have appropriately discussed the roentgen anatomy of the various systems of the body and have provided brief descriptions of disease processes as they concern the radiologist. Although this book is designed largely for the radiologist, it provides an excellent reference for the practicing physician, surgeon, and other specialists in medicine. It will serve as one of the finest text books that is available in the English language for the undergraduate medical student.

The authors have considered all of the common conditions observed in the United States, and many of the uncommon ones. They have included and discussed all of the methods of examinations used in modern radiologic techniques. They have exhibited an unusual ability to record accurately and concisely the important aspects of the various conditions.

The type is easy to read and the illustrations have been carefully chosen and are beautifully reproduced. A fairly sizable bibliography is included which provides a ready reference library.

This volume should serve not only the young radiologists, but it should be most acceptable to experienced radiologists. The organization of the material in the volume has been according to systems and in that way one finds, readily available, a brief and concise description of most of the common conditions encountered in the practice of radiology.

The publishers deserve a great deal of credit for the calender of the paper, the excellence and the size of the illustrations and the excellent type used in the manuscript. This reviewer is most enthusiastic and recommends it highly.

Eugene P. Pendergrass, M.D.

A Text-Book of X-Ray Diagnosis by British Authors in Four Volumes. Volume IV. Third edition. Edited by S. Cochrane Shanks, C.B.E., M.D., F.R.C.P., F.F.R., Director, X-Ray Diagnostic Department, University College Hospital, London; and Peter Kerley, C.V.O., C.B.E., M.D., F.R.C.P., F.F.R., D.M.R.E., Director, X-Ray Department, Westminster Hospital; Radiologist, National Heart Hospital, London. Cloth. Pp. 714, with 735 illustrations. W. B. Saunders Company, 218 West Washington Square, Philadelphia, Pa., 1959.

For twenty-two years this text has been one of the standard reference works in the field of radiology. Much progress has been made since the second edition was published in 1950, and the present text has kept abreast of the advances with up-to-date references and source material. The general format is unchanged. Illustrations of roentgenograms continue to be positive prints of the negative films, which many in this country find less familiar.

Volume IV is again devoted entirely to the roentgenology of bones, joints and soft tissues, with one chapter discussing localization of foreign bodies. In addition to Drs. Shanks and Kerley there are nineteen distinguished contributors, each a specialist in his own field of radiology. Emphasis is not placed on the rare or theoretical disease; the material is geared to the current practice of diagnostic roentgenology.

Those who have used this well-known text in the past will most certainly wish to bring their reference shelf up-to-date with this volume.

Arch H. Hall, M.D.

Radiographic Atlas of Skeletal Development of the Hand and Wrist. Second edition. By William Walter Greulich, Professor of Anatomy, Stanford University School of

Medicine; and S. Idell Pyle, Research Associate, Department of Anatomy, Western Reserve University and Stanford University Schools of Medicine. Cloth. Price, $15.00. Pp. 256, with numerous illustrations. Stanford University Press, Stanford, Calif., and Oxford University Press, London, 1959.

The second edition of this atlas is an indispensable addition to the library of all who are concerned with the roentgenographic assessment of skeletal age. Although no dramatic changes have been made in comparison with the previous edition, an excellent chapter by Bayley and Pinneau has been added which discusses the prediction of maximal adult height from the skeletal age as determined in the atlas. The reproductions are excellent.

This volume lucidly presents extensive information necessary for the refinement of one's ability to evaluate skeletal age.

George R. Barnes, Jr., M.D.

Atlas of Roentgenographic Measurement. By Lee B. Lusted, M.D., Associate Professor of Radiology, University of Rochester School of Medicine and Dentistry; and Theodore E. Keats, M.D., Professor of Radiology, University of Missouri School of Medicine. Cloth. Price, $9.00. Pp. 176, with 119 illustrations. The Year Book Publishers, Inc., 200 East Illinois Street, Chicago, Ill., 1959.

This is a very useful collection of carefully selected measurement data covering almost every field of medicine with which the radiologist is concerned. The material has been obtained from the most reliable medical literature of approximately the last two decades.

A unitized collection of standards such as this has been sorely needed by radiologists for years. Those who have kept a drawer full of reference notes to various articles in a host of different medical journals will know this to be true. Many radiologists keep a miscellaneous assortment of celluloid covered tables and charts which sometimes become lost or mislaid. One may not fully agree with the selection of source material in every case but, in general, this book should please even the most opinionated practitioner.

Chapters are devoted to each of the major body organ systems, presenting average organ dimensions and positions. Included are measurements of cerebral ventricles, soft tissue thicknesses in the neck, pelvimetry using the Ball and Colcher-Sussman methods, average kidney, spleen, and adrenal gland sizes, biliary and gastrointestinal average dimensions and the important heart and great vessel indices. The well known Ungerleider tables are reproduced. The largest portion of the text is devoted to skeletal measurements and maturation. Every measurement is illustrated in atlas-like detail, and technical constants are given in each case. Most radiologists should find this book an indispensable addition to their reference shelf.

Arch H. Hall, M.D.

Atlas of Roentgenographic Positions. Volumes I and II. Second edition. By Vinita Merrill. Cloth. Price, $32.50. Pp. 635, with numerous illustrations. C. V. Mosby Company, 3207 Washington Boulevard, St. Louis 3, Mo., 1959.

This new edition, made necessary by the advances in roentgenography during the last ten years, has a concise, readable text and many excellent illustrations.

The first volume, after a general introduction to the subject and a description of the preliminary steps in roentgenography, discusses general anatomy and anatomic terms, and the anatomy and positioning of the upper and lower extremities, the shoulder girdle, the bony thorax, the pelvic girdle, and the vertebral column. A glossary of medical terms and an extensive bibliography conclude this volume.

The second volume deals with the anatomy of the skull and positioning of the head, the paranasal sinues, pneumoencephalography and cerebral angiography, positioning for study of the facial bones, anatomy and positioning for study of the teeth and salivary glands, neck, body cavities, and digestive, urinary and genital systems. An extensive bibliography is included also in this volume.

Because of the abundance of the illustrations, the excellent drawings, and the clear, concise exposition, the reviewer feels that this work is valuable both as a teaching text and as a reference book. The author and the publisher have done a real service in providing a second and more complete edition of a book which was already a classic in its field.

C. Gianturco, M.D.

Die Wirbelsäulenleiden und ihre Differentialdiagnose. By Prof. Dr. J. E. W. Brocher, Genf. Cloth. Price, $30. 50. Pp. 457,

with 287 illustrations. Georg Thieme Verlag, Herdweg 63, Stuttgart, Germany, 1959. In the U.S.A. and Canada, Intercontinental Medical Book Corporation, New York 16, N. Y.

Prof. Brocher has drawn on his own obviously extensive and scholarly experience plus the (Western) European and American literature for the second edition of his monograph. While the major emphasis is on diagnosis, particularly roentgenologic, pathogenesis and treatment are also briefly discussed. The excellent illustrations, in almost every instance, take the form of succinct case presentations; hence, the 287 "illustrations" consist of 639 separate reproductions and diagrams. The examples illustrated and the discussions in the text deal more with situations met early in the ordinary course of events than with the "classical," full-blown (or worse) examples of vertebral abnormalities.

He deliberately minimizes discussion of vertebral fractures. Vertebral tuberculosis and other infections, ankylosing spondylarthritis, Scheuermann's disease, spondyloses, and the congenital abnormalities get extended discussions, as do primary tumors and diseases which cause decreased density, increased density (or both), or altered osseous structure. Separate chapters deal with the sacrum and with differential diagnostic points. Twenty pages of references and an index complete the work.

Prof. Brocher writes that roentgen techniques which are certainly reproducible must be adapted to each patient, and he makes a strong case for teleroentgenography, roentgenography with the patient upright, and laminagraphy.

The style of the monograph is free and easy despite being concise; the text is both to the point and yet is happily near poetic in some of its descriptions. This monograph should be studied by radiologists, orthopedic surgeons and general practitioners.

Henry G. Moehring, M.D.

Evaluation of the Pelvis in Obstetrics. Second edition. By Charles M. Steer, M.D., Med.Sc.D., F.A.C.S., F.A.C.O.G., Associate Professor of Clinical Obstetrics and Gynecology, College of Physicians and Surgeons, Columbia University, and the Sloane Hospital for Women. Cloth. Price, $4.00. Pp. 131, with 57 illustrations. W. B. Saunders Company, 218 W. Washington Square, Philadelphia, Pa., 1959.

Originally written by the late Dr. Howard Moloy, this book now appears in a second, revised version prepared by Dr. Charles Steer who collaborated with Dr. Moloy in studies to determine means of recognizing the important obstetric problem of cephalopelvic disproportion. It is a small, concise volume with excellent illustrations and a highly readable text. For the obstetrician and the radiologist the material presented here is an excellent guide in situations involving dystocia originating in the bony pelvis.

The book is divided into five sections, the first two of which deal with the general morphology of the pelvis based upon the well-known classification and criteria of Caldwell and Moloy, and the techniques of clinical examination of the pelvis. Section III describes the mechanism of labor and the importance of pelvic shape in determining the adaptation of the head at the inlet in gynecoid, android, anthropoid and platypelloid pelves. Section IV discusses the significance of pelvic shapes as they relate to techniques of delivery in mid-pelvic and low medium arrest in transverse, posterior, and anterior positions of the fetal head. Section V deals with the recognition of disproportion by clinical and roentgenologic techniques and with attempts to determine whether pelvic delivery is not merely possible but, more important, safe for the child.

This last chapter, of particular interest to readers of these pages, describes roentgenologic techniques and criteria developed by the authors and their associates at the Sloane Hospital. Degrees of disproportion were expressed by differences between the diameter of the circle which fits the inlet and the diameter of the fetal head. By many correlations with the outcome of labor, these differences provided "an accurate statistical prognosis" on the likelihood, though not absolute assurance, of delivery from below in any individual case. Even with such limitations the approach outlined in this small volume demonstrates that a satisfactory measure of precision in obstetric pelvimetry is presently available in situations where formerly only clinical intuition was the deciding factor.

Harold C. Mack, M.D.

La Stratigrafia dei Bronchi. By L. Oliva; R. Vignolini; and G. L. Besio. Paper. Price, L.4000. Pp. 242, with 61 illustrations. Edizioni Minerva Medica, Torino, Italy, 1959.

The authors have employed laminagraphy in

the study of bronchial pathology very extensively and this monograph is a summary of their experiences.

The first part is devoted to the various laminagraphic techniques which can be employed in the study of the bronchial tree and to the laminagraphic anatomy of the bronchi. The second part deals with the laminagraphic findings in congenital, inflammatory and neoplastic diseases.

A comparison between bronchography and laminagraphy of the bronchi closes the book. According to the authors, laminagraphy has the main advantage of being the simpler of the two examinations. It will reveal pathology in bronchi which cannot be filled by the opaque medium, and will allow the study of peribronchial tissue. On the other hand, bronchography shows much better detail in that part of the bronchial tree which can be filled.

The authors conclude that in many cases both techniques should be employed to arrive at a satisfactory understanding of the underlying pathology.

C. Gianturco, M.D.

Analytical Cytology; Methods for Studying Cellular Form and Function. Second edition. Edited by Robert C. Mellors, M.D., Ph.D., Pathologist, Director of Laboratories, and Associate Director of Research, Hospital for Special Surgery, Philip D. Wilson Research Foundation, an Affiliate of the New York Hospital-Cornell Medical Center, New York; Formerly at the Sloan-Kettering Institute for Cancer Research and the Memorial Center for Cancer and Allied Diseases. Cloth. Pp. 534, with numerous illustrations. The Blakiston Division, McGraw-Hill Book Company, Inc., 575 Madison Ave., New York 22, N. Y., 1959.

This book is written by seven authorities in this field of scientific investigation, which encompasses biophysical and biochemical cytology. The theory and practical applications of these methods of analysis of cellular structure and function are fully discussed. Detailed accounts of the following research techniques are given: fluorescent-antibody method; intracellular localization of chemical constituents, phase, interference, and polarization; electron and roentgen-ray microscopy; autoradiography and photometric chemical analysis of cells. The text is concise and to the point, so that even a novice could readily understand and apply these techniques.

This book will stimulate the reader into thinking about the nature of the cellular constituents and will arouse the investigative impulse of any researcher. So many problems remain unanswered and this book serves as a guide into the field. The black and white photographs are well reproduced and the print is easy to read. The bibliography at the end of each chapter is up to date, complete and comprehensive.

The reviewer recommends this book to all those interested in medical research, whether they be technicians, teachers or graduate students, and to those working in closely allied fields of medical science.

T. Jarkowski, M.D.

An Introduction to Electronics for Physiological Workers. Second edition. By I. C. Whitfield, B.Sc., Ph.D., Reader in Physiology, University of Birmingham. Cloth. Price, $3.75. Pp. 263. Macmillan and Co. Ltd., London; St. Martin's Press, Inc., 103 Park Ave., New York 17, N. Y., 1959.

The subject is covered in the range between elementary and more advanced radio text books for graduate students. This is written from the biologist's viewpoint broadly conceived as physiology, botany, pharmacology, agriculture, zoology and medicine. The book is beautifully illustrated with numerous small diagrams. There is a pleasing contrast between the dull surface of the paper and the clear black ink. The size also helps make the mechanics of reading easy. It is well indexed. The author accomplished his purpose very effectively.

The book achieves admirably the task expected from the title.

Walter H. Seegers, Ph.D.

High Altitude and Satellite Rockets. Foreward by A. D. Baxter, Chairman of the Organizing Committee. Cloth. Price, $15.00. Pp. 136, with numerous illustrations. The Philosophical Library, Inc., 15 East 40th Street, New York 16, N. Y., 1959.

This is a record of a symposium held at Cranfield, England, July 18–20, 1957, and sponsored by The Royal Aeronautical Society, The British Interplanetary Society and The College of Aeronautics. The core of the volume is its technologic information with diagrams,

charts, photographs, and extensive references. There are twelve presentations, and a statement issued by Prof. Boris M. Petrov of the U.S.S.R. Academy of Sciences on the U.S.S.R. rocket and earth satellite program for the International Geophysical Year. There is a short biography of each author and the discussions are recorded.

Walter H. Seegers, Ph.D.

Roentgens, Rads, and Riddles; A Symposium on Supervoltage Radiation Therapy, Held at the Medical Division, Oak Ridge Institute of Nuclear Studies, July 15, 16, 17, and 18, 1956. Edited by Milton Friedman, M.D., Marshall Brucer, M.D., and Elizabeth Anderson. Cloth. Price, $3.50. Pp. 495, with numerous illustrations. United States Atomic Energy Commission, 1959. For sale by the Superintendent of Documents, Government Printing Office, Washington, D. C.

This is an obviously strenuously edited transcript of a seminar gathered in 1956 at, if not officially under the auspices of, the Medical Division of the Oak Ridge Institute of Nuclear Studies for the stated purpose of provoking discussions on supervoltage therapy by some 75 invited guests. As was inevitable, the work turns out to be a sort of omnium-gatherum in the general area of the stated subject. It confirms the observation that when participants from various disciplines, with experiences ranging from extensive to anticipatory, partake in extended conversations on a subject, they often operate on the general principle observed by some duck hunters that, if enough guns are shot often enough in the general direction of the objective, and for a long enough period of time, something of merit will be brought down. Quite a few birds of a considerable variety were brought down, all of which were apparently not headed in the same direction, and quite a few feathers as well.

The book is divided into five general parts. The first deals with "Dosage Considerations" and is written largely by physicists. It is the best of the lot and contains a very lucid description of the meaning and use of the roentgen and the rad. Part II deals with "Apparatus and Sources." It presents in almost 100 pages the various gadgets which a number of physicians, physicists, and private and commercial agencies were thinking about making, were making, had made, or were selling in 1956, and still are. Part III is a collection of 32 presentations under the general title of "Principles of Supervoltage Irradiation." There are four subsections entitled "Moving-Fields Therapy," "Clinical Radiobiology," "Indications and Contra-Indications," and "Special Techniques for Special Tumors." Part IV contains the transcript of three "clinics" under the general title "Discussions of Case Problems." The discussions in these two sections show evidence of a spread in experience ranging from considerable, critical and sophisticated to "one case which was followed for six months." Part V, "Special Talks," contains several interesting philosophical discussions on trends in radiotherapy and gives opportunity for the editors to include subjects dear to their hearts but not quite germane to the title, "Brachytherapy Devices." The volume ends, as befits a symposium held at the Medical Division of ORINS, with a gentle plug for cesium 137 as a teletherapy source.

There is a lot of good information buried in this book. The lack of an index may have the salutory effect of forcing the reader to peruse the entire book in order to find it. It is hardly to be used as a ready reference, but rather as a source of general reading by those about to be newly confronted by or otherwise concerned with the problems peculiar to supervoltage. It does indicate the status and direction of supervoltage radiotherapy in 1956 and it makes good reading in the more general field of radiation therapy.

The riddle is not solved, but acquaintance with the unit is made. It being, of course, the unit which explains (?) why tumor plus patient plus rads never comes out to a constant.

The book is very well constructed and organized; the topography is excellent and the illustrations and diagrams profuse and clear. It is fortunate that it was published by the United States Government Printing Office. Had it been privately or commercially printed, the price of $3.50 would have been impossible. As it is, at $00.00686 per text page, it is a good buy.

Milford D. Schulz, M.D.

BOOKS RECEIVED

Röntgenanatomie. By Prof. Dr. med. D. Nagy, Vorstand des Institutes für chirurgische Anatomie und experimentelle Chirurgie der medizinischen Universität, Budapest. Cloth. Pp. 509, with 545 illustrations. Akadémiai Kiadó, Budapest, 1959.

Radiation Biology. Proceedings of the Second Australasian Conference on Radiation Biology held at the University, Melbourne, 15–18 December, 1958, by The Australian Radiation Society. Edited by J. H. Martin. Cloth. Price, $11.00. Pp. 304, with numerous illustrations. Academic Press Inc., Publishers, 111 Fifth Ave., New York 3, N. Y., 1959.

Safe Handling of Radioactive Isotopes in Medical Practice. By Edith H. Quimby, Sc.D., Professor of Radiology (Physics), College of Physicians and Surgeons, Columbia University. Cloth. Price, $4.50. Pp. 129. The Macmillan Company, 60 Fifth Ave., New York 11, N. Y., 1960.

Genetics and Cancer. A Collection of Papers Presented at the Thirteenth Annual Symposium on Fundamental Cancer Research, 1959. Edited by the Staff of The University of Texas M. D. Anderson Hospital and Tumor Institute. Cloth. Price, $8.50. Pp. 459, with numerous illustrations. The University of Texas Press, Austin 12, Texas, 1960.

Roentgen Examinations in Acute Abdominal Diseases. Second edition. By J. Frimann-Dahl, M.D., Ph.D., Chief of the Roentgen Department, Ullevål Hospital, Oslo, Norway. Cloth. Price, $15.50. Pp. 518, with 446 illustrations. Charles C Thomas, Publisher, 301–327 East Lawrence Ave., Springfield, Ill., 1960.

Encyclopedia of Medical Syndromes. By Robert H. Durham, M.D., F.A.C.P., Physician-in-Charge, Division of General Medicine, Henry Ford Hospital, Detroit. Cloth. Price, $13.50. Pp. 628. Paul B. Hoeber, Inc., Medical Book Department of Harper & Brothers, 49 East 33rd St., New York 16, N. Y., 1960.

Cancer and Allied Diseases of Infancy and Childhood. Edited by Irving M. Ariel, M.D., Associate Professor of Clinical Surgery, New York Medical College; Attending Surgeon, Hospital for Joint Diseases; Staff Surgeon, Long Island Jewish Hospital; Attending Surgeon, Pack Medical Group, New York, N. Y.; and George T. Pack, M.D., Associate Professor of Clinical Surgery, Cornell University Medical College; Attending Surgeon, Memorial Center for Cancer and Allied Diseases; Attending Surgeon, Pack Medical Group, New York, N. Y.; and 27 Contributing Authors. Cloth. Price, $22.50. Pp. 605, with numerous illustrations. Little, Brown & Company, 34 Beacon St., Boston 6, Mass., 1960.

Outline of Human Genetics. By L. S. Penrose, M.A., M.D., F.R.S., Galton Laboratory, University College, London. Cloth. Price, $2.50. Pp. 146. John Wiley & Sons, Inc., 440 Fourth Ave., New York 16, N. Y., 1959.

Intussusception in Infants and Children. Edited by Mark M. Ravitch, M.D., Associate Professor of Surgery, The Johns Hopkins University School of Medicine; Surgeon-in-Chief, The Baltimore City Hospitals, Baltimore, Md. Cloth. Price, $9.00. Pp. 121, with numerous illustrations. Charles C Thomas, Publisher, 301–327 East Lawrence Ave., Springfield, Ill., 1959.

ABSTRACTS OF RADIOLOGICAL LITERATURE

Department Editor: T. Leucutia, M.D., Harper Hospital, Detroit 1, Michigan

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ROENTGEN DIAGNOSIS

Neck and Chest

Gondos, Bela, Hunter, Charles H., and Braunwald, Nina. Dissecting aneurysm of the aorta; report of two cases diagnosed by angiocardiography. *New England J. Med.*, Sept. 10, 1959, *261*, 535–539. (From: Department of Radiology, District of Columbia General Hospital, Washington, D. C.)

With the recent development of techniques for the surgical correction of dissecting aneurysm of the thoracic aorta, the early diagnosis of this entity assumes great importance. The clinical impression is often sufficient to raise the suspicion that this condition is present. Conventional roentgenography can demonstrate a dissecting aneurysm only if serial roentgenograms show progressive widening of the aortic shadow or, occasionally, if the presence of intimal calcification marking the inner wall of the aorta permits the demonstration of increased thickness of the aortic wall.

Angiocardiography is an excellent means of establishing a diagnosis of dissecting aneurysm by demonstration of irregular narrowing of the main aortic ahannel and widening of the space between the outer margin of the channel and the outer margin of the aortic outline. Less commonly, the false channel is demonstrated by the opaque medium.

The authors report 2 cases in which the clinical diagnosis of dissecting aneurysm was confirmed by angiocardiography. The first patient underwent succcessful surgery and was asymptomatic twenty-five months after operation. The second patient showed spontaneous clinical and roentgenographic improvement, presumably due to rupture of the aneurysm into the lumen of the aorta, and was asymptomatic nine months after the onset of the disease.—*Leslie K. Sycamore, M.D.*

Dunbar, J. Scott, and Favreau, Martine. Infrapulmonary pleural effusion with particular reference to its occurrence in nephrosis. *J. Canad. A. Radiologists*, June, 1959, *10*, 24–33. (From: Montreal Children's Hospital, Montreal, Quebec, Canada.)

The history of the accepted concept of pleural effusion localization is briefly discussed in the opening paragraphs. Reference is made to Rigler's first article in 1931, in which he reported 2 cases of "atypical" collections of fluid in the chest. The fluid formation was described as overlying the diaphragm and it had a convex upper surface, simulating elevation of the diaphragm.

Infrapulmonary pleural effusions are now readily recognized. However, there has not been a great deal of interest in this type of effusion occurring in nephrosis. The authors' interest was aroused by a high incidence of infrapulmonary pleural effusions in a group of 75 patients who had been seen in their hospital since 1948. The chest roentgenograms and records of 49 of these patients who had had chest roentgenograms made during the active stage of the nephrosis were reviewed. Of these cases, 36 were thought to show evidence of fluid between the base of the lung and the diaphragm. This is about 75 per cent of the patients who had suitable roentgenograms made during the active stage of the nephrosis.

A list of the signs believed to be of importance in the identification of the fluid is given and each sign is discussed and illustrated.

The authors conclude that infrapulmonary pleural effusion can be recognized by any one of the following changes: (1) reverse meniscus with an appearance of a depressed cardiac position when there is bilateral fluid formation; (2) change in contour of the lung base with the curve of the lung base reaching its maximum height at approximately the junction of its middle and lateral thirds rather than at its center; (3) fluid in the costophrenic sinus posteriorly; (4) separation of the stomach bubble from the left leaf of the diaphragm; (5) displacement of fluid with posture change, usually best demonstrated on roentgenograms in the lateral decubitus position; and (6) a fairly characteristic left lower triangular mediastinal fluid collection, which is best seen on slightly overexposed roentgenograms.

The authors note that their survey of the previous literature shows only 8 case reports of patients with nephrosis and infrapulmonary pleural effusion. They believe, after reviewing their own material, that the association of fluid formation and active nephrosis is the rule rather than the exception; if this fluid formation is looked for it can be found in almost every case of active nephrosis.

The illustrations presented in this article are excellent.—*Richard E. Kinzer, M.D.*

Baker, David H., Maisel, Bernard, and Goldberg, Henry P. Use of pneumoperitoneum in differential diagnosis of paracardiac masses in children. *A.M.A. Arch. Surg.*, July, 1959, *79*, 63–67. (Address: D. H. Baker, New York Hospital, 525 E. 68th St., New York 21, N. Y.)

The authors report 3 cases of paracardial masses in children. These were investigated by roentgenograms made following induction of a pneumoperitoneum. In each of these cases, a small eventration of the diaphragm was demonstrated on the right side with herniation of a small portion of the liver into it.

The authors suggest that this procedure may frequently be of help in studying masses of this nature. —*George L. Sackett, Jr., M.D.*

Levin, Bertram. Subpleural interlobular lymphectasia reflecting metastatic carci-

noma. *Radiology*, May, 1959, *72*, 682–688. (Address: Michael Reese Hospital, 29th St. and Ellis Ave., Chicago 16, Ill.)

Subpleural interlobular lymphatic dilatation was first described by Kerley in 1951, when he reported the occurrence of these lines in silicosis and attributed them to lymphectasia. The dilated lymphatics appear on the chest roentgenogram as short transverse lines in the bases of the lungs, 1 to 2 cm. long, extending outward to the pleura. They vary in number from few to many and are arranged in step-ladder fashion. They are usually bilateral but occasionally unilateral and are generally confined to the lower half of the lung fields. Kerley's opinion has since been verified and the occurrence of these lines has been reported in cases of mitral valve disease, pneumonia, and in other conditions with impaired pulmonary lymphatic drainage. Basal septal lines have been encountered as a prominent roentgenologic feature in many cases of carcinomatous metastases in the lungs, in some instances being the first clue that a malignant lesion was present.

Five representative cases with chest roentgenograms are presented to illustrate the roentgen appearance, though this does not differ from that in cases of other etiology.—*Walter H. Jarvis, Jr., M.D.*

Shields, Thomas W., and Anderson, Marion C. Thoracic esophageal diverticula; report of three cases. *Am. J. Digest. Dis.*, July, 1959, *4*, 522–533. (From: The Department of Surgery, Northwestern University Medical School, 303 E. Chicago Ave., Chicago 11, Ill.)

The authors discuss the problem of intrathoracic esophageal diverticula in a simple and clear fashion. They consider diverticula either epibronchial or epiphrenic according to their point of origin in the intrathoracic esophagus. Pathogenesis, symptomatology, complications, and methods of diagnosis of each are presented.

The epibronchial or traction diverticulum is the most common variety and rarely produces symptoms.

The epiphrenic diverticula are of infrequent occurrence and only 126 cases were published in the literature up to 1952. Since that time an additional series of 121 pulsion (epiphrenic) diverticula seen at the Mayo Clinic has been reported. These diverticula arise 5 to 7.5 cm. proximal to the cardia and the majority project to the right. They are considered to be pulsion in origin though traction may play a role once the pouch has developed. The herniation may occur through a congenitally weak area in the muscular wall, but the factors which initiate the herniation are not completely understood.

Three cases are reported with typical gallbladder symptoms and findings which proved to be secondary to epiphrenic diverticula instead of cholecystitis. Surgery is the treatment of choice for diverticula which are symptomatic or large in size.—*Raul Gonzales, M.D.*

Abdomen

Gütgemann, A., and Parchwitz, H. K. Ösophagus- und Magenvarizen und ihr röntgenologischer Nachweis. (Esophageal and gastric varicosities and their roentgenologic demonstration.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, May, 1959, *90*, 547–558. (From: Chirurgische Universitäts-Klinik, Bonn, Germany.)

Since 20 to 50 per cent of patients with liver cirrhosis die of bleeding from esophageal and gastric varices, their demonstration is of great significance in view of prognostic evaluation and therapeutic measures. Considering the anatomy and physiology of the portal circulation, one has to expect greater distention of the short gastric veins of the fornix than those of the paraesophageal venous plexuses. The fact, however, that, roentgenologically, the gastric varicosities are found less frequently than the esophageal has its explanation in the great variability of tonus and function of the fornix, and in the different submucosal structure. With improvement of the examination technique the number of positive findings in the stomach has increased markedly and in the reports of 1953, about 25 per cent of patients with esophageal varices were shown roentgenographically to have gastric varicosities.

Special technical procedures are aimed at full barium filling of the fundus as well as double contrast study by positioning and gas distention, using carbonated water if necessary and taking multiple rapid spot roentgenograms while changing from the horizontal to the upright position. Following the routine examination, particular consideration is given to the size and shape of the liver and spleen, as well as to the pattern of the small bowel loops for persistence of edema of the folds. The normal mucosal pattern of the fornix is characterized by a curly appearance with the concavities of the arcades directed downward. In the body portion of the cardia the mucosal folds assume a parallel course. This normal pattern is, however, subject to great variations commensurate with the shape, tonus, and filling capacity of the stomach. In fornix varicosities one finds a row of filling defects up to the size of a cherry, with wavy demarcation of the contours. This appears against the air and barium shadow as an increased soft tissue density in the cardia portion of the fornix. Such a case is presented, showing markedly dilated venous plexuses of the cardia and lower esophagus with a clearly demonstrated filling defect due to thrombus in the portal phlebogram. Four weeks after the portocaval anastomosis a significant regression of the varicosities was noted.

Frequently, visualization of fornix varicosities is made difficult by the presence of a considerable degree of spleno- or hepatomegaly compressing the fornix. This difficulty can be overcome by air distention of the cardia—a procedure which is especially helpful in evaluating the therapeutic results of portocaval shunts. Actually, absence of gastric varicosities with proved esophageal varicosities is very uncommon. More frequently, the opposite is true, *i.e.*, the presence of large gastric varicosities with few esophageal varices. However, if the esophageal varicosities are extremely large, it is possible to have gastric varicosities which are only slightly developed. The explanation may be that in the narrow space of the esophagus the varicose-forming elements can exert greater pressure on the contrast material than in the large space of the fornix.—*Nathan Kriss, M.D.*

Holle, F., and Viehweger, G. Ösophagealer Reflux nach subdiaphragmatischer Fundektomie; seine klinische und röntgenologische Erkennung und seine Vermeidung. (Esophageal reflux after subdiaphragmatic fundus resection; its clinical and roentgenologic recognition and prevention.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, May, 1959, *90*, 564–572. (From: Chirurgische Universitätsklinik, Würzburg, Germany.)

Normally, the esophageal reflux is prevented by the following factors: (1) the oblique entrance of the esophagus into the stomach; (2) the sharp angle between the esophagus and the greater curvature, the so-called Hissch angle which acts as a valve; (3) the Guba valve; (4) the air bubble in the cardia which increases pressure toward the esophageal lumen and acts with the Guba valve as a pneumatic shut off; (5) the oblique muscle fibers in the cardia area contracting the Hissch angle and accentuating the cardia incisura; and (6) the limbs of the diaphragmatic hiatus acting as a muscle sling around the lower esophagus. The disturbance of this muscle by atony or interruption leads to displacement of the cardia and ineffectiveness of the previously active control mechanisms. The tonus in the lower esophagus and cardia of the stomach is the regulating agent. Lack of one or more of these factors may lead to reflux esophagitis.

Sixteen of 46 cases of fundectomies for cardia ulcers and fundus carcinoma were intensively investigated, clinically and roentgenologically, for the presence of esophageal reflux or reflect esophagitis. Approximately half of the patients with roentgenologically proved reflux had no complaints. It is, therefore, not surprising that mucosal changes rarely could be demonstrated. The patients were divided into three groups. In the first group (3 cases) there was no reflux and completely normal passage. In the second group (8 cases) some reflux was observed in the Trendelenburg position and in the right lateral oblique projection but no reflux was seen in the upright position. In the third group (5 cases) very severe reflux was found in the upright position during normal swallowing. Functional results of surgery were good in the first and second groups and reflux was absent in all those cases in which a satisfactory restitution of normal cardia anatomy was achieved.

The following points should be carefully observed in performing the operation: (1) the anastomosis must definitely be in clear sight with widely opened abdomen; (2) the anastomosis should never enter at the highest point of the stomach stump but be placed end-to-side after situating the stump on the anterior wall; (3) the implantation of the esophagus should be made on the anterior wall of the stomach stump between the upper and middle third and in an oblique line from left up to right down; (4) the serosal and mucosal sutures at the posterior wall of the stomach stump near the esophagus should be placed apart in such a way as to form a Guba valve; (5) the new-formed fundus should be fixed with sutures to the left diaphragm; (6) denervation must be avoided.

All cases showed a temporary initial inflammatory and edematous mucosal swelling at the anastomosis. It was noted that this swelling receded much sooner in carcinoma cases than in ulcer patients. In 2 of these, a stenosis developed, caused by reflux esophagitis, which receded after dilatation and the local application of cortisone. Extensive resection of the fundus, unavoidable in some cancer patients, produced more persistent and difficult reflux complaints. This led the authors to abandon fundectomy in fundus carcinomas in favor of total gastric resection by the abdominothoracic procedure with interposition of a small bowel loop between the esophagus and the duodenal stump. This placement of a sufficiently long innervated reservoir underneath the esophagus provides a very effective mechanism for the prevention of reflux (Nakayama-Longmire operation). Where the fundus can be sufficiently restituted, the preservation of at least half of the normal gastric volume and the reduction of gastric stasis by pyloromyotomy is considered an adequate procedure.—*Nathan Kriss, M.D.*

Hamrick, Leon C. Gastric perforation in the newborn infant. *J.A.M.A.*, Sept. 26, 1959, *171*, 411–414. (Address: 700 Ridgeway Rd., Fairfield, Ala.)

In the 3 cases reported in this article, the lesion was unrecognized in 1 patient until autopsy and a second patient died after surgery from duodenal perforation thought to be caused by prolonged intragastric intubation and unrelieved associated duodenal obstruction. The third infant survived after corrective surgery and is developing normally.

Early diagnosis and prompt surgical treatment are

necessary in order to reduce the high mortality associated with this condition.—*Peter C. Truog, M.D.*

de Larrechea, Ignacio, Schapira, Adolfo, Ramos Mejia, Manuel M., and D'Alotto, Victorino. Valor de los signos radiologicos del sindrome de malabsorcion. (Value of the radiologic signs of the malabsorption syndrome.) *Prensa méd. argent.*, Jan. 16, 1959, *46*, 178–185. (Address: I. de Larrechea, Libertad 1199, Buenos Aires, Argentina.)

Ten cases of sprue and 1 case of celiac disease were studied in order to evaluate the roentgen findings in the malabsorption syndrome. Roentgenograms of the abdomen were made at sixty minute intervals for five hours after administration of 250 cc. of barium mixture. Evidence of dilatation of the small intestine associated with segmentation was present in 54.5 per cent; segmentation was found in 81.0 per cent. All of the cases studied showed hypersecretion and alteration of the intestinal folds. No consistent findings were observed in the transit time. The moulage sign was found in only 5 cases. Using the above criteria, roentgen diagnosis could be made in 54.5 per cent of the cases of sprue.

Clinical improvement was obtained with steroids, folic acid, vitamins and diet.—*Murray M. Friedman, M.D.*

Rubin, Sidney, and Dann, David S. Pseudotumors of the colon. *G. P.*, Sept., 1959, *20*, 102–105. (From: Department of Radiology, University of Kansas Medical Center, Kansas City, Kan., and Menorah Medical Center, Kansas City, Mo.)

Colon deformities demonstrated fluoroscopically and roentgenographically may be due to adherent mural feces. In the first such case encountered by the authors, the colon appeared adequately cleansed and the lesion was persistent fluoroscopically and roentgenographically even after evacuation and refilling and fluoroscopic manipulation of the area. At operation no lesion was found and repeat barium enema study postoperatively revealed a normal colon.

A fecal mass may attach itself to the mucosa of the colon in such a cohesive way that the most energetic fluoroscopic manipulation along with the barium pressure fails to detach it. Any defect which could be attributed to this, therefore, should be checked by re-examination.—*Leslie K. Sycamore, M.D.*

Nelson, William A. and Rogers, James V., Jr. Lipoma of the colon: with report of seven cases. *South. M. J.*, July, 1959, *52*, 767–773. (From: The Department of Radiology, Emory University School of Medicine, Atlanta, Ga.)

Lipoma is the second most frequent benign tumor occurring in the colon. Although relatively rare, more than 200 cases have been reported in the literature. The authors have reviewed 7 cases observed in their institution since 1949.

Lipomas occur more frequently in the colon than anywhere else in the digestive tract. Although in most previous series about one half of the lipomas of the colon occurred in the cecum and ascending colon, all 7 cases presented here occurred in the distal part of the colon.

Lipomas are usually solitary tumors which most often arise in the submocosa, but they may be subserosal, intermuscular or intramuscular in location. They may be pedunculated or sessile. None have been known to undergo malignant change. About 80 to 85 per cent are symptomatic. The symptoms consist of constipation, intermittent abdominal pain often relieved by the passage of flatus or a bowel movement, and, sometimes, constipation alternating with diarrhea. Intestinal obstruction, intussusception, and ulceration with bleeding are not infrequent complications. These tumors are occasionally expelled spontaneously.

The roentgenographic appearance is fairly characteristic. The tumors are usually smooth, rounded and quite large, sometimes measuring as much as 10–15 cm. in their greatest diameter. This is in contrast to the ordinary adenomatous polyp, which is small and verruciform or berry-like in appearance. Lipomas have a tendency to assume an oval or sausage configuration due to their pliability which permits them to mold in conformity with the lumen of the bowel. This pliability can sometimes be demonstrated on spot roentgenograms made under pressure. Occasionally, on air contrast study, they are more radiolucent than one would expect with a tumor of their size, due to their fat content.

It is interesting to note that, in the 7 cases studied by the authors, 2 of the patients spontaneously expelled the tumor and in 3 of the 7 cases the diagnosis was suspected from the roentgen findings. In 1 of the cases intussusception occurred and the authors concluded that the presence of intussusception in an adult should make one suspect the possibility of a lipoma of the colon. Practically all of the cases presented the characteristic complaints and showed the rather typical roentgenographic changes outlined above.—*Russell F. Scalf, M.D.*

Rogers, C. William. Lipomas of the colon; report of a case in a patient with multiple neurofibromatosis (von Recklinghausen's disease). *Radiology*, May, 1959, *72*, 754–756. (Address: Department of Radiology, University of Chicago, Chicago 37, Ill.)

The case history of a patient with colonic lipomas causing intussusception and intermittent obstruction, with associated multiple neurofibromatosis, is

presented. Neurofibromatosis is classified as one of the congenital ectodermoses, sometimes called neurocutaneous syndromes, which include tuberous sclerosis, angiomatosis cerebri, and Lindau-von Hippel's disease. Visceral tumors are a common occurrence in tuberous sclerosis.

In the several hundred reports of colonic lipoma in the literature, no specific mention was found of a patient with von Recklinghausen's neurofibromatosis. It is the view of the author, however, that the finding of visceral lipoma in this case may suggest a close relationship between the neurocutaneous diseases in that the findings typical of one syndrome may be present in another.—*W. M. McBride, M.D.*

Kourias, B., and Tobler, A. Das Postcholecystektomie-Syndrom; auf Grund unserer Spätergebnisse bei 551 nachuntersuchten Fällen der Jahre 1950–1955. (The postcholecystectomy syndrome; on the basis of our late findings in 551 followed-up cases from 1950 to 1955.) *Chirurg.*, Sept., 1959, *30*, 398–403. (From: Chirurgische Klinik des Krankenhauses von Griechischen Roten Kreuz, Athens, Greece.)

In order to investigate the postcholecystectomy syndrome, the authors reviewed the results of cholecystectomies done by them between 1950 and 1955 in 551 patients. Of these, 69.5 per cent had a cholecystectomy only; 26 per cent also had a choledochotomy. Of these latter, 93.5 per cent had external drainage and 6.5 per cent had a choledochoduodenostomy. The percentage of re-operation was low (2.51 per cent).

The authors believe that their favorable results are probably due to systematic use of operative cholangiography. Since 1950 they have done 1,400 operative and postoperative cholangiographies. No detail is given regarding their technique and findings in this extensive series.—*H. W. Hefke, M.D.*

Thal, Alan P., Goott, Bernard, and Margulis, Alexander R. Sites of pancreatic duct obstruction in chronic pancreatitis. *Ann. Surg.*, July, 1959, *150*, 49–56. (From: Departments of Surgery and Radiology, University of Minnesota, Minneapolis, Minn.)

In chronic pancreatitis, the pancreatic duct may be obstructed in one or several areas within the substance of the pancreas at points distant from the papilla of Vater. Treatment aimed at relief of obstruction requires precise localization of the site of obstruction.

Sometimes satisfactory pancreatograms may be obtained during cholangiography, especially if the papilla is made spastic by instillation of 0.1 *N* hydrochloric acid into the duodenum. Direct pancreatography can be performed during laparotomy by introduction of a polyethylene catheter (4-F) through the pancreatic duct into the tail of the pancreas. This fine catheter will pass through most strictures. Small amounts of 38 per cent renografin are allowed to reflux from the tail of the gland into the duodenum.

Eight cases are reported exhibiting obstruction at various sites. In 2 cases of acute pancreatitis, obstruction was revealed at the ampulla of Vater during the attack. In 1 case of chronic pancreatitis, obstruction was demonstrated at the ampulla and was relieved by sphincterotomy. In another case of chronic disease, there was complete obstruction at the ampulla with dispersion of the opaque material in the gland substance, which has been described as a sign of chronic pancreatitis. A case of obstruction of the pancreatic duct proximal to the ampulla in the head of the pancreas, was relieved by pancreatic jejunostomy. In a patient where multiple strictures were demonstrated, a pancreaticojejunostomy could not be accomplished because of fixation of the pancreas to the posterior gastric wall, but a fistulous tract from the pancreas was anastomosed to the stomach. The patient did well for twelve months, at which time he developed a fistulous tract draining externally. Two additional cases of chronic disease showed obstruction at the papilla and in the head of the pancreas in 1 instance, and in the head of the gland in another; both were relieved by decompression.

Since in the chronic stage the disease responds less satisfactorily to treatment, localization of the site of the obstruction and correction during the early stages of the disease is important.—*Lois Cowan Collins, M.D.*

Evans, John A., and Mujahed, Zuheir. Roentgenographic aids in diagnosis of neoplasms of liver and extrahepatic ducts. *J.A.M.A.*, Sept. 5, 1959, *171*, 7–11. (Address: J. A. Evans, 525 E. 68th St., New York, N. Y.)

The histories of 35 patients with primary carcinoma of the gallbladder were reviewed in order to assess the diagnostic value of preoperative roentgenography. An upper gastrointestinal series was performed in 32 of these patients and in 10 of them there were signs of extrinsic pressure on the duodenum. A plain roentgenogram of the abdomen revealed a mass in the right upper quadrant in 3 of 24 cases. Oral cholecystography was carried out in 26 cases, 4 of which showed opacification of the biliary system. The authors do not mention whether or not the above roentgenographic findings enabled the radiologist to make the correct diagnosis preoperatively in any of the patients.

Operative cholangiography is occasionally of value in the diagnosis of bile duct neoplasms, especially in the intrahepatic ducts, which are inaccess-

ible to palpation. Intravenous cholangiography has not been of value in the diagnosis of primary neoplasm of the biliary tract.

Percutaneous transhepatic cholangiography may provide a definitive diagnosis in neoplasms of the biliary tract. The technique is not without certain risk and the authors have restricted its use to the immediate preoperative period.—*J. L. Williams, M.D.*

Taylor, W. Jape, Jackson, F. C., and Jensen, Wallace N. Wilson's disease, portal hypertension and intrahepatic vascular obstruction. *New England J. Med.*, June 4, 1959, *260*, 1160–1164. (From: Departments of Medicine and Surgery, University of Pittsburgh School of Medicine, and the Veterans Administration Hospital, Pittsburgh, Pa.)

The authors investigated the portal circulation of 4 patients with classic Wilson's disease (hepatolenticular degeneration).

Hepatic vein catheterization was done with measurement of pressures in the distal and central hepatic veins. Hepatic blood flow was calculated. Splenic puncture with pressure measurements and subsequent splenic portograms were done.

There was portal hypertension in all patients, but no evidence of extrahepatic obstruction. The hepatic blood flow in these cases was either normal or reduced. Thus, the portal hypertension must be the result of intrahepatic vascular obstruction.

The extrahepatic portal bed is patent, but the wedged hepatic vein pressure is not elevated. An adequate collateral system must be interposed between the site of intrahepatic obstruction and the catheter wedged in the hepatic vein.

The authors suggest that the site of vascular obstruction in Wilson's disease is at the presinusoidal level of intrahepatic circulation.—*George L. Sackett, Jr., M.D.*

Douglas, F. Clark, and Bailey, Herbert A. (Dallas, Tex.) Gastrografin: in gastroenterologic diagnosis. *South. M. J.*, Sept., 1959, *52*, 1003–1008.

Gastrografin is a 76 per cent aqueous solution of the salt of diacetylamino-triiodobenzoic acid. It has low toxicity, is completely soluble in water and is miscible with body fluids. Only a small amount is absorbed from the intestinal tract. It is readily absorbed from the peritoneal cavity and it does not appear to be toxic to the tracheobronchial tree.

Four ounces of undiluted gastrografin are given orally for study of the upper gastrointestinal tract. Visualization of the esophagus is good. In the stomach and duodenum there is adequate visualization in most instances but lack of adhesiveness makes it possible to overlook small mucosal lesions. In comparison with barium, craters are not visualized as easily and some may be missed. The material demonstrates retention and obstruction and is able to pass through high degrees of obstruction more readily than barium. Gastrografin does not give satisfactory contrast for detailed study of the small intestine because it becomes diluted. It has an unusually rapid transit through the small intestine and usually reaches the cecum in thirty to sixty minutes, often outlining the colon in one to three hours. Given by enema, it is not as satisfactory as barium for studying the colon.

The outstanding advantage of gastrografin is the absence of impaction and inspissation in the intestinal tract. This factor makes it desirable for use in cases of suspected obstruction in any part of the gastrointestinal tract. It is of value in the examination of patients with problems of constipation and especially in cases of known or suspected megacolon.

Disadvantages of gastrografin include its low contrast in the small intestine, an unpleasant taste, high cost, and a definite laxative effect.—*J. L. Williams, M.D.*

Woodruff, J. H., Jr., Ottoman, R. E., Simonton, J. H., and Averbrook, B. D. The radiological differential diagnosis of abdominal trauma. *Radiology*, May, 1959, *72*, 641–650. (Address: J. H. Woodruff, Jr., Harbor General Hospital, Torrance, Calif.)

A total of 159 cases of abdominal trauma on whom roentgen examinations were performed prior to surgery have been reviewed and analyzed. Each case was reviewed in its entirety, with history, physical, laboratory, roentgen and surgical findings all being correlated in the analysis. Types of injuries were classified as nonpenetrating or penetrating, and with single or multiple sites of involvement.

The amount of diagnostic work performed for each patient varied, depending on the patient's condition and the type, site, and extent of injuries. Flat, upright and decubitus roentgenograms of the abdomen and an upright chest roentgenogram were obtained when possible. Other special procedures were carried out when necessary. These included cystography, urography, retrograde pyelography and aortography. Blood cell counts and urinalysis were done routinely when possible. Four quadrant taps to search for fluid were performed on 33 patients.

The structures most commonly injured were the spleen, abdominal wall, small intestine, liver, bladder and kidney. Those giving a high incidence of positive findings on the roentgenograms were the bladder, kidney and spleen. Intra-abdominal fluid, when seen on the roentgenograms, was due, in about half the cases, to a splenic injury. This was also a not uncommon finding in injuries of the liver, bladder, mesenteric and other extravisceral vascular structures. The visualization of an enlarged left upper quadrant mass was highly specific for splenic injury but was liable to occasional false negative and false positive inter-

pretations. The demonstration of a right upper quadrant density larger than the normal liver shadow was infrequent. In only 2 of 11 patients in whom the injury was confined to the liver was this finding observed. Pneumoperitoneum was found only 9 times and retroperitoneal emphysema only once in the entire series. Out of 29 penetrating wounds, with no perforation of a hollow viscus, only 2 showed free air in the abdomen. Patients who were found at surgery to have perforations of gastrointestinal tract organs and who had satisfactory preoperative roentgenograms numbered 27. Of this group 7 showed pneumoperitoneum and one retroperitoneal emphysema. These findings reveal that pneumoperitoneum, when present, was a generally reliable indication of perforation of a hollow viscus but that its absence was of little significance. Retroperitoneal hematoma, identified by obliteration of the psoas muscle and by a local increase in density, was due in half of the cases to kidney injuries. The next most common cause was splenic trauma, with mesenteric artery and liver injuries being an occasional cause.

In general, splenic, bladder and kidney injuries resulted from nonpenetrating trauma, while intestinal tract, abdominal wall, diaphragm, gallbladder and pancreatic injuries usually were due to a penetrating type of trauma. Liver, mesenteric and extravisceral vascular structure injuries were due to either nonpenetrating or penetrating trauma. With nonpenetrating injuries, the site of external trauma generally was over the site of internal injury. Multiple site injuries were difficult to evaluate but frequently the roentgen studies aided in establishing the diagnosis. —*Donald N. Dysart, M.D.*

Genitourinary System

Ross, James A. One thousand retrograde pyelograms with manometric pressure records. *Brit. J. Urol.*, June, 1959, *31*, 133–140. (From: Edinburgh Royal Infirmary and Leith Hospital, Edinburgh, Scotland.)

The author reports the results of 1,000 retrograde pyelographic examinations using a mercury manometer in circuit to observe and control the pressure. In this series, when 5 to 6 ml. of contrast medium had been injected, the average pressure was 53.6 mm. Hg. In the first 880 injections with local anesthesia of the urethra, 5.3 per cent complained of discomfort. The subsequent examinations were made with the addition of an antispasmodic drug, buscopan, injected into the renal pelvis, and discomfort occurred in only 1.7 per cent. Complaints occurred at variable pressure readings, as low as 15 mm. Hg in 1 case but not even as high as 100 mm. Hg in another.

Pyelorenal backflow took place in 2.7 per cent of the injections. Twenty-four of the 27 instances of backflow occurred in normal kidneys, being unrelated to renal pathology. The lowest pressure at which it was noted was 15 mm. Hg; the average was 50.2 mm. Hg. In 10 cases there was some accompanying discomfort but in 15 no discomfort was noted. The 2 patients who were under general anesthesia did not complain of discomfort afterwards. It was concluded that the threshold for pain and discomfort is as variable in the kidney as it is elsewhere in the body in different persons.

On account of the variable pressures at which discomfort and backflow occurred, it is not possible to define a critical level for the development of these complications. However, the manometer in circuit provides a valuable indicator, warning the examiner to keep the pressure within moderate limits. This is particularly important when general anesthesia is used.—*Arthur E. Childe, M.D.*

Rubin, E. L., Ross, J. Cosbie, and Turner, D. P. B. Cystic disease of the renal pyramids ("sponge kidney"). *J. Fac. Radiologists*, July, 1959, *10*, 134–137. (From: Royal Southern Hospital and Broadgreen Hospital, Liverpool, England.)

Clinically, patients with cystic disease of the renal pyramids usually present with a mild, intermittent urinary infection, easily controlled, but tending to recur. The roentgenologic picture is characteristic of the condition, appearing as cystic spaces (filled with contrast medium) contiguous with most, or all, of the terminal calices. The appearance has been described as resembling clusters of grapes around each calyx. It is seen on excretory urography, but not, as a rule, on retrograde examination. If stones form, they are located in the small paracalyceal cysts and, if multiple, they may resemble nephrocalcinosis. Usually both kidneys are affected.

In 2 cases reported by others in which nephrectomy was performed, the removed kidney was slightly enlarged. The cysts were confined to the medulla. They varied in diameter from the size of a pinhead to a pepper corn, but an occasional larger cavity resulting from the confluence of several cysts was found. The cysts were dilatations of the collecting tubules and were lined by flattened cuboidal or low columnar epithelium.

The prognosis in this disease is excellent. The common complications are infection and the deposition of calcium to form small concretions in the paracalyceal cysts.

The two conditions listed by the authors as necessary for consideration in the differential diagnosis are renal tuberculosis and the chronic form of necrosis of the renal papillae, in which there may be remissions and exacerbations extending over a period of several years.—*Samuel G. Henderson, M.D.*

Payne, Richard A. Clinical significance of reduplicated kidneys. *Brit. J. Urol.*, June, 1959, *31*, 141–149. (From: Urological De-

partment, United Sheffield Hospitals, Sheffield, England.)

A series of 141 cases of reduplicated kidneys has been reviewed. In 28 cases the presenting symptom was pain, either on the side of the reduplication or in the low back. Forty-four patients presented with symptoms of urinary tract infection. Hematuria and pyrexia were occasionally found. In 41 cases the symptoms could not definitely be ascribed to the reduplication. Calculi were present in 10 patients, usually in the lower segments. Occasionally, there were other anomalies such as spina bifida occulta, hypospadias and multiple anomalies of the urinary tract.

The author considers that reduplication of the upper urinary tract may be the basic anomaly responsible for some cases of enuresis, due to a persistent urinary infection or, rarely, an ectopic ureter in the female. Sometimes the diagnosis is difficult when there is a nonfunctioning atrophic element, and both excretory and retrograde pyelograms may be necessary. The ten minute retention roentgenogram in retrograde pyelography is an important part of the investigation.

Treatment in this series has been conservative but nephrectomy or heminephrectomy is sometimes indicated.—*Arthur E. Childe, M.D.*

Cordonnier, Justin J. Unilateral renal artery disease with hypertension. *J. Urol.*, July, 1959, *82*, 1–9. (Address: 4960 Audubon Ave., St. Louis, Mo.)

The author presents 7 cases of unilateral renal artery disease. These cases illustrate various lesions such as congenital narrowing of the renal artery, partial renal infarction with renal ischemia and atherosclerosis of the main renal artery.

On the basis of the findings on intravenous urography and clinical study, three classifications of etiology for unilateral hypertension are evident: (a) narrowing of the main renal artery with no passage of opaque medium on excretory urography; (b) infarction with some remaining but ischemic parenchyma; (c) narrowing of the renal artery with normal intravenous pyelogram.

Intravenous pyelography is of definite value as a screening procedure in all cases of hypertension and the presence of diminished function or decrease in the size of one kidney may be of some significance. However, it is pointed out that this finding alone is not sufficient basis for nephrectomy. In 2 of the author's cases, the dye excretion was bilaterally adequate, but the aortogram in these instances showed evidence of renal artery disease; therefore, normal intravenous pyelograms do not rule out the possibility of unilateral vascular disease. Differential sodium studies and volume excretion studies of urine are also of value as a screening procedure but the results must be substantiated by aortography.

In those patients who have typical clinical evidence of renal infarction, or in those who have no demonstrable function of one kidney on intravenous urography, and in whom differential function studies indicate unilateral arterial disease, aortography is not recommended, since there is a certain degree of hazard associated with it. In these clear-cut cases, surgery is indicated without aortography.

Results of surgical treatment by nephrectomy in the cases presented have been gratifying and indicate the need for a more thorough evaluation of hypertensive patients in regard to unilateral renal artery disease.—*George W. Chamberlin, M.D.*

Howell, Robert D. Milk of calcium renal stone. *J. Urol.*, Aug., 1959, *82*, 197–199. (Address: 1802 N. Illinois St., Indianapolis 2, Ind.)

This is a single case report of a thirty-seven year old white female with urinary tract infection. She was found to have a calcific density lying in the renal parenchyma adjacent to the middle calyx of the left kidney, as visualized roentgenographically in both the anteroposterior and lateral views. This was at first thought to be a calcium stone, but it could not be found at operation. Subsequent roentgen studies following surgery showed that in the lateral decubitus and erect positions there was a fluid level produced by the calcified density. The homogeneous quality of this material suggested a similarity to milk of calcium, such as is seen in the gallbladder.

This case illustrates the value of the erect position as a routine procedure in the roentgen study of the abdomen in these patients.—*George W. Chamberlin, M.D.*

Rene, Robert M., and Mellinkoff, Sherman M. Renal insufficiency after oral administration of a double dose of cholecystographic medium; report of two cases. *New England J. Med.*, Sept. 17, 1959, *261*, 589–592. (From: Department of Medicine, University of California School of Medicine, Los Angeles Medical Center, Los Angeles, Calif.)

The authors have observed 2 cases of renal insufficiency following the administration of a double dose of iopanoic acid. The first patient was a forty-two year old male who was given a double dose (6.0 gm.) of iopanoic acid because of a previous inconclusive examination with a single dose. On the following morning during a cholecystographic examination, he had an episode of orthostatic hypotension after which he had a period of oliguria with associated elevation of serum creatinine and blood urea nitrogen. These reached a peak on the seventh day after administration of the medium with a gradual fall to normal by the twenty-first day. He suffered no aftereffects and was well when last seen nineteen months later.

The second patient was a sixty year old female who was given a double dose of iopanoic acid because of nonvisualization of the gallbladder at previous examination with a single dose of bunamiodyl. Twelve hours later she complained of inability to void. Sixty ml. of dark brown urine was obtained upon catheterization. The following day she complained of pain in the lower abdomen radiating to the left flank and costovertebral angle as well as frequency and burning on urination. The abdominal symptoms disappeared with bed rest and a low fat diet. Blood chemistry studies showed elevation of serum creatinine and blood urea nitrogen with peak values of 14.5 mg. creatinine on the eighth day and 95.8 mg. urea nitrogen on the twelfth day. Normal values were restored five weeks after she had received the iopanoic acid.

There has been no previous report of renal insufficiency following the administration of iopanoic acid. Renal excretion of the medium amounts to approximately 38 per cent of the administered dose and dysuria is not an uncommon symptom. Renal irritation in susceptible persons may result in reflex vascular spasm with partial or complete renal shutdown. It seems wise not to administer large doses of gallbladder dye to patients with evidence of preexisting renal disease or to do so, if necessary, only after the possible risk has been carefully weighed.—*Leslie K. Sycamore, M.D.*

Pasquier, C. M., Jr., St. Martin, E. C., and Campbell, J. H. (Shreveport, La.) Further observations on the problems of vesicoureteral reflux. *South. M. J.*, Aug., 1959, *52*, 967–974.

This is a study of vesicoureteral reflux in 500 children twelve years of age or younger. Cystograms, including postvoiding roentgenograms, were made. Reflux was found in 14 per cent of cases and most of these were females (83 per cent).

The authors review the theories as to the cause of reflux and agree with the concept that it usually follows intravesical obstruction or neurogenic dysfunction, and that there are changes in the bladder wall which permit the intravesical portion of the ureter to become extravesical and thereby result in reflux. They believe that the bladder neck obstruction is more common than usually realized and they have several criteria to identify this on cystoscopic examination. The occurrence of reflux should lead one to make a diligent search for a cause.

Patients with a mild degree of reflux and with a correctable cause, such as bladder neck obstruction, usually do not require plastic repair of the ureterovesical junction, and have no further trouble following correction of the basic difficulty. Cases with severe reflux, however, continue to have trouble unless repair is done to prevent the continued reflux.

Decision as to the necessity for repair of the ureterovesical junction in cases of intermediate severity is quite difficult. The authors have tried to evaluate these cases with cystometric studies, but accurate correlation is not easy. They feel that, if changes are present in the upper urinary tract along with reflux, repair of the ureterovesical junction is probably indicated to prevent further reflux and kidney damage.—*George L. Sackett, Jr., M.D.*

Shaheen, David J., and Johnston, Andrew. Bilateral ureteral obstruction due to envelopment and compression by an inflammatory retroperitoneal process: report of two cases. *J. Urol.*, July, 1959, *82*, 51–57. (Address: 258 Genesse St., Utica, N. Y.)

The authors report 2 patients, a sixty-one year old female and a forty-eight year old male, who illustrate a rather unusual cause of bilateral ureteral obstruction. Since 1948 when Ormond first published an instance of this disease, there have been 17 such cases reported in the literature. It now appears that this condition is being recognized more frequently. The etiology is unknown, and the disease usually appears without any previous history that would suggest its cause. Some of the means which have been used to relieve the obstruction are: (1) ureterolysis with nephrostomy; (2) antibiotic therapy; (3) radiation therapy; and (4) combinations of these procedures.

The roentgen findings, as demonstrated by intravenous or retrograde pyelograms, are those which are usually associated with lower ureteral obstruction. In the 2 cases presented in this article, there was no visualization of the left kidney by intravenous pyelography and the right side showed evidence of hydronephrosis. Surgery revealed the ureteral obstruction to be due to a well-formed "sleeve" of inflamed periureteral tissue which enveloped the ureters from the pelvic brim to the ureteropelvic junction.

The authors suggest the possibility of phlebitis which may originate in the ovarian or spermatic veins as a possible etiologic factor.—*George W. Chamberlin, M.D.*

Gerlaugh, Robert L., Rattner, William H., and Murphy, John J. Late results of urethroplasty for stricture. *J. Urol.*, June, 1959, *81*, 763–769. (From: Department of Surgery, Division of Urology, Hospital of the University of Pennsylvania and the Harrison Department of Surgical Research, School of Medicine, University of Pennsylvania, Philadelphia, Pa.)

A common factor in all strictures is the presence of infection in and around the narrowed area. The diagnosis can be suspected clinically and can be made with certainty by the use of urethrography.

This permits accurate localization of the diseased area as well as other valuable information regarding its extent and severity. Thixokon has been found to be a satisfactory medium because of its consistency and radiopaque quality. This is injected into the urethra through a Brodney clamp, and roentgenograms are made in the anteroposterior and oblique bicycle riding position following the injection of approximately 20 to 30 cc. of opaque medium.

The authors describe in detail the technique for surgical repair of strictures of the anterior and of the posterior urethra. The principles are based upon open drainage of infection in and around the urethral stricture until complete healing has occurred. This is followed by a reconstruction of the urethra by various technical methods. The clinical course of the patients operated on indicates that the method is satisfactory.—*George W. Chamberlin, M.D.*

Davidson, J. K., Harvard, C. W. H., and Scott, R. Bodley. Radiological demonstration of enlarged retroperitoneal lymph-nodes. *Lancet*, May 16, 1959, *1*, 1008–1010. (From: St. Bartholomew's Hospital, London, E. C. 1, England.)

Demonstration of enlarged retroperitoneal lymph nodes becomes a problem in many instances because they are difficult to palpate. Frequently, blind irradiation of a large abdominal field is necessary in treating cases of Hodgkin's disease. In order to eliminate unnecessary treatment, the authors have investigated the value of retroperitoneal pneumography as a more accurate means of determining whether enlarged lymph nodes are present.

Their technique is described in great detail. The patient is prepared as for excretion pyelography. Preliminary roentgenograms and laminagrams are taken. He is then placed in the right lateral recumbent position and a lumbar puncture type needle is inserted a little to one side of the midline and midway between the anus and coccyx. The needle is introduced 4–6 cm. and 5 or 10 cc. of normal saline solution is injected. The saline solution collects around the point of the needle. Aspiration is attempted to make certain that the needle does not lie within a blood vessel. The pool of fluid about the point of the needle is thought to prevent gas embolism. During the next five to seven minutes, 600 ml. of oxygen is slowly insufflated. The patient is then turned on the opposite side and the procedure is repeated, making a total insufflation of about 1,200 ml. of oxygen. Following this, he is allowed to sit up and walk around the room for fifteen to twenty minutes, then roentgenograms are taken. The authors state that no complications have occurred in 38 patients whom they examined by this method.

A description of the positive findings follows and there are several good illustrations showing the pertinent findings. It is agreed that lesions less than 4 cm. in diameter are probably not demonstrated.

The authors believe that demonstration of an otherwise undetectable lesion allows for better treatment. They also believe that wide field irradiation is often not necessary when a definite diagnosis can be arrived at by this method.—*Richard E. Kinzer, M.D.*

Scanlan, David B. Primary retroperitoneal tumors. *J. Urol.*, June, 1959, *81*, 740–745. (From: Department of Urology, Atlantic City Hospital, Atlantic City, N. J. and Shore Memorial Hospital, Somers Point, N. J.)

Primary retroperitoneal tumors are those which arise independently of any organ in the retroperitoneal area. They may arise from ectocerm, mesoderm or mixed cell types, and their rate of malignancy is high.

The author has compiled 688 cases of primary retroperitoneal tumors from the literature. One hundred and fifty-two of these were benign and 536 were malignant. The cell type is noted in the table of these tumors.

In another table included in this article, the operability rate, as noted by six different investigators, is found to be as low as 7.4 per cent and as high as 89.9 per cent depending upon the individual reporting the cases. The average operability is approximately 30 per cent, but in the individual patient this depends upon the nature of the lesion encountered. Of all the primary retroperitoneal tumors, the most common one is that which arises in fat cells, namely, the lipoma or liposarcoma.

The author reports a case of a man with a 19½ pound liposarcoma which was removed at operation. Two other case histories given are those of a lymphosarcoma and a primary epithelial tumor.—*George W. Chamberlin, M.D.*

Abeshouse, George A., Goldstein, Robert B., and Abeshouse, Benjamin S. Adrenal cysts: review of the literature and report of three cases. *J. Urol.*, June, 1959, *81*, 711–719. (From: Department of Urology, Sinai Hospital, Baltimore, Md.)

Cysts of the adrenal gland are rare lesions which present difficult diagnostic problems until they have reached a large size. In this article the authors have collected 152 instances of adrenal cysts from the literature and have added 3 cases personally operated by them. These cysts will occur at any age, but are most commonly observed in the fifth and sixth decades of life. They may occur on either the right or left side, and they are three times more common in the female than in the male.

The most common type of adrenal cyst is the pseudocyst, of which there were 51 cases reported in the series. This type of cyst is the end result of hemor-

rhagic extravasation in or around the adrenal gland or in a tumor of the adrenal gland. Such cysts may show evidence of calcification within their walls.

The second most common variety of adrenal cysts is the endothelial cyst, which is derived from degeneration of lymphatic and vascular structures. They have been variously described as cystic lymphangiectasia, hygroma, retention cysts, and struma medullaris cystica.

The less common types of cysts noted in this particular series are parasitic cysts, congenital retention cysts, and cystic adenoma. Many of these lesions are asymptomatic and are found incidentally on routine autopsy.

The three most common clinical features are dull pain in the adrenal area, gastrointestinal symptoms and a palpable mass. When the diagnosis is suspected, roentgenographic studies including intravenous urography, laminagraphy, retrograde pyelography and retroperitoneal air injection are indicated. The presence of calcification and a cystic mass in the retroperitoneal region is strongly suggestive of an adrenal cyst. That it is not always located above the kidney is illustrated by 1 of the authors' patients. The lesion in this particular instance displaced the renal pelvis and upper third of the ureter ventrally and laterally, but the cyst apparently arose from the adrenal gland and extended retroperitoneally, dorsal to the kidney and to the upper ureter.—*George W. Chamberlin, M.D.*

Nervous System

Drewes, J., and Gremmel, H. Neurogene Tumoren der Lungen. (Neurogenic tumors of the lungs.) *Thoraxchirurgie*, July, 1959, *7*, 40–51.(From: Chirurgische Klinik und Institut für Medizinische Strahlenkunde der Medizinischen Akademie, Düsseldorf, Germany.)

Most neurogenic tumors in the thorax originate from nervous tissue outside of the lung, that is, from the spinal cord or sympathetic nervous system. Neurogenic tumors arising in the lung tissue itself are rather rare. Only 30 such cases could be found in the more recent literature.

Five cases of neurogenic tumors of the lungs observed in the Surgical Clinic of Düsseldorf are reported.

The symptoms and clinical findings were in no way specific. Roentgenologically, the tumors were well circumscribed, round or oval in shape. Bronchography sometimes showed displacement of the bronchial tree; a bronchial obstruction was found only once. Three of the 5 tumors were neurofibromas, 1 was a neurinoma, and the other was a neurilemmoblastoma with malignant sarcomatous degeneration. Pneumonectomy, lobectomy and segmental resection were the surgical procedures.—*Hans W. Hefke, M.D.*

Schweisguth, O., Mathey, J., Renault, P., and Binet, J. P. Intrathoracic neurogenic tumors in infants and children: a study of forty cases. *Ann. Surg.*, July, 1959, *150*, 29–41. (From: The Institut G. Roussy Villejuif [Seine] and the Clinic of Thoracic Surgery and Laboratory of Pathology, Hospital Laënnec, Paris, France.)

The authors review 40 cases of intrathoracic neurogenic tumors in children. These are classified: neuroblastoma, 15 cases; ganglioneuroblastoma, 7 cases; and ganglioneuroma, 18 cases.

In all neuroblastoma cases but 1, there was resection of all or part of the tumor followed by radiation therapy with 1,500 to 3,000 r tumor dose in from twenty-two to forty-five days. In some cases, preoperative therapy was given. Although follow-up was short (six months to seven years), there were 6 children considered as probable cures; 5 had received postoperative radiation and 5 of the 6 were under the age of two years. One case was well four years after incomplete removal and no roentgen therapy.

The authors believe that surgery alone is adequate for infants under the age of two with neuroblastoma. In children over the age of two years, because of the poorer prognosis, surgery plus radiation therapy should be used.

In the 7 ganglioneuroblastomas, treatment varied and could not be evaluated. The authors recommend postoperative radiation therapy.

The ganglioneuromas were all successfully handled by surgery alone.—*Henry J. Klos, M.D.*

Kvernland, Bruce N., Grewe, Ray, V., Woolley, Ivan M., and Lee, J. Robert. Upright large volume dynamic myelography. *Radiology*, April, 1959, *72*, 562–568. (Address: B. N. Kvernland, Emanuel Hospital, Portland 12, Ore.)

Pantopaque myelography as it is usually carried out, using 6 to 9 cc. of contrast medium with the patient in the prone, oblique and lateral positions, has been shown to be inadequate for the demonstration of many types of neurologically significant lesions of the spine. This is especially true of lesions causing encroachment on the posterior surface of the spinal canal. Pantopaque, being heavier than spinal fluid, gravitates into the anterior compartment of the canal when the patient is in the prone position. Even when the patient is brought into a semi-upright or upright position, only a small segment of the posterior canal surface is actually outlined. In addition to this, many lesions may become evident only during weight-bearing or bending motions of the spine and cannot be demonstrated when the patient assumes the prone position. This fact is frequently substantiated by the clinical history.

Grayson and Black as well as others have tried to

eliminate some of these deficiencies by increasing the amount of contrast material used in order to fill the canal more completely. Bell *et al.* have appraised the effect of motion and position upon the contours of the canal, and Brierre and Colclough used the combination of a large volume of medium plus weight-bearing in their study. The authors of the present study have combined all four factors: large volume of medium, weight-bearing, motion, and position. The technique is described in detail. The salient features are as follows: (1) Volume of the medium used may be as much as 36 cc. and as little as 12 cc., the average being 16–18 cc. (2) The larger volumes are used in cases with high lesions or with large neural canals. (3) Routine myelograms are taken in the prone, oblique and lateral projections with the patient in a slightly head-up position. (4) The patient is then elevated into a standing position on the footboard. (5) He is then seated on a tall stool and positioned in a true lateral projection. (6) Extension and flexion myelograms are taken with the tube centered at the level of the iliac crest. (7) He is returned to the table and lowered into a semi-Fowler's position and the medium is aspirated.

This technique is recommended primarily for patients who display definite cauda equina type symptoms. Included in this group are those who have had solid fusions of L-4, L-5 and the sacrum without relief of symptoms and those in whom inadequate fusion occurred and symptoms persisted. Others included are those in whom routine myelography was done and a lesion was not found to explain the clinical and neurologic findings.

Four case histories are presented along with excellent reproductions of roentgenograms to demonstrate the comparative value of the technique.—*Z. Petrany, M.D.*

Skeletal System

Sullivan, Walter E., and Miles, Meryl. The lumbar segment of the vertebral column. *Anat. Rec.*, April, 1959, *133*, 619–636. (From: University of Wisconsin, Madison, Wis.)

The authors studied the vertebral column of 49 young adults and conclude that there is some degree of lateral curvature and torsion normally found in the lumbar segment. The curvature and torsion may be major, minor or minimal. In lateral roentgenograms several types were seen varying from an almost straight segment with a dorsal inclination to marked curvature approaching a lordosis. There was 1 instance of only four free vertebrae in the lumbar segment, a female, and 3 with six free vertebrae, 2 females and 1 male.—*Peter C. Truog, M.D.*

Dega, Wiktor, Król, Jerzy, and Polakowski, Lech. Surgical treatment of congenital dislocation of the hip in children; a one-stage procedure. *J. Bone & Joint Surg.*, July, 1959, *41-A*, 920–934. (From: The Orthopaedic Clinic of the Medical Academy, Poznán, Poland.)

A one stage operative procedure for congenital dislocation of the hip in children has been used by the authors and their clinic since 1953. The method entails: (1) a capsular arthroplasty or acetabuloplasty, (2) an oblique subtrochanteric and subperiosteal derotation osteotomy, and (3) a femoral shortening osteotomy. This one stage method shortens the period of immobilization to a minimum. It is used as a primary treatment in neglected cases in which treatment is started late, and as a secondary operation in cases previously treated unsuccessfully.

Any one or all of the steps in the procedure may be used if indicated in a given case. Capsular arthroplasty is used when a disproportion exists between the acetabulum and the femoral head. Femoral shortening is done when there is high upward displacement of the epiphysis, the procedure affording muscular relaxation and preventing undue pressure of the femoral head against the acetabulum. A derotation is indicated if the femoral neck and head are anteverted more than 45 degrees. Osteotomy to correct a valgus deformity of more than 145 degrees is done to prevent deterioration and overloading of the femoral head.

The preliminary follow-up study (two to four years) suggests that the most favorable age for this procedure ranges from three to five years. Of all children in this age group, 94 had good or excellent results. Failures are apt to occur in children over the age of six years due to vascular changes which develop despite correct operative technique.—*Arch H. Hall, M.D.*

Ponseti, Ignacio V., and Frigerio, Edgar R. Results of treatment of congenital dislocation of the hip. *J. Bone & Joint Surg.*, July, 1959, *41-A*, 823–846. (From: Department of Orthopaedic Surgery, State University of Iowa, Iowa City, Iowa.)

The importance of early diagnosis and treatment in congenital dislocation of the hip can never be too strongly stressed. Results of a review of a very large series with minimal follow-up periods of four to eight years showed that all patients initially treated before the age of one year had good anatomic results. Good results dropped to 78 per cent in infants one to two years of age, and they decreased to only 57 per cent in those two to four years of age.

The only unequivocal indication of early dislocation is Ortolani's snapping sign. The displaced femoral head snaps into the center of the acetabulum when the greater trochanter is pressed forward and the thighs abducted. This occurs in all preluxations, but it is absent in subluxations where definitely positive roentgenographic findings are present. True dis-

locations are, of course, recognized both clinically and roentgenographically. However, in young infants a sloping acetabular roof and delay in ossification of the femoral head is compatible with normal development of a hip in infants with a negative Ortolani sign.

The dislocations were reduced under general anesthesia and only 3 open reductions were done when closed reduction failed. The authors believe that closed reduction, followed by carefully supervised treatment, is the method of choice if the child is seen before he is four years of age.—*Arch H. Hall, M.D.*

Gram, Peter B., Fleming, Joseph L., Frame, Boy, and Fine, Gerald. Metaphyseal chondrodysplasia of Jansen. *J. Bone & Joint Surg.*, July, 1959, *41-A*, 951–959. (From: The Departments of Orthopaedic Surgery, General Medicine, and Pathology, Henry Ford Hospital, Detroit, Mich.)

In 1934 Jansen reported an unusual case of disturbed endochondral bone formation which he termed metaphyseal dysostosis. The features consisted of gradual progressive swelling of the joints and stunted growth; metaphyses were enlarged and still cartilaginous at the age of ten; spots of irregular calcification and ossification were scattered throughout the metaphyses; the diaphyses were curved and thickened with irregular and expanded ends; serum calcium ranged from 13 to 16.6 mg. per cent.

The condition is extremely rare and the authors describe only the fourth such case in world literature. The roentgenologic features included: (1) large, irregular, clear areas of uncalcified cartilage adjacent to the epiphyseal line and under the thin cortical margins of the expanded metaphyses; (2) patchy, irregular, denser areas representing calcified cartilage in the metaphyses; (3) areas of irregular ossification near the diaphyses; and (4) wide irregular epiphyseal lines with relatively normal epiphyses.

Biochemical changes of importance were persistent hypercalcemia, hypophosphatemia, elevated serum alkaline phosphatase, increased urinary excretion of calcium and phosphorus, and lowered renal tubular resorption of phosphorus. These biochemical findings were similar to those found in hyperparathyroidism, but roentgenologic skeletal findings were at variance with this condition. The epiphyseal cartilage ossified, diaphyseal density was normal, and a marked disturbance in epiphyseal growth occurred. However, in this case the parathyroid glands were surgically explored and no detectable abnormality was found.

The authors have differentiated the condition from hypophosphatasia, hypervitaminosis D, vitamin D resistant rickets, renal tubular acidosis, Fanconi's syndrome, Ollier's disease, chondrodystrophia calcificans congenita, and hereditary metaphyseal dysplasia. They have suggested the name "metaphyseal chondrodysplasia of Jansen" as appropriate for this abnormality, which is essentially a persistence of epiphyseal cartilage and a defect in the formation of endochondral bone. It is postulated that the metabolic changes—hypercalcemia and hypophosphatemia—may have been secondary to the underlying bone defect.—*Arch H. Hall, M.D.*

Moulonguet, P., and Cazala, J.-Fr. (Paris, France.) Chondromes évolutifs sur exostoses ostéogéniques. (Growing chondromas from osteogenic exostoses.) *Presse méd.*, May 27, 1959, *67*, 1055–1057.

Seven cases are presented and the subject is summarily reviewed. The condition is not as rare as it formerly was thought to be. Most of these cases are benign, arising from small foci of cartilaginous cells at the periphery of or within the exostoses. A few have a locally invasive and recurrent character. Fewer still are frankly malignant (some with metastasis). These latter are, as a whole, less malignant than other malignant tumors of bones.

The authors refer to the recent book by Jaffe on bone tumors and seem to concur with his conclusion that chondrosarcomatous (peripheral) degeneration of solitary exostoses is rare (1 per cent) in contrast to more frequent malignant changes (25 per cent) of the familial type of exostoses.—*Jirair N. Sarian, M.D.*

Stein, Arthur H., Jr. Benign neoplastic and non-neoplastic destructive lesions in the long bones of the hand. *Surg., Gynec. & Obst.*, Aug., 1959, *109*, 189–197. (From: Orthopedic Division of the Department of Surgery, Washington University School of Medicine, St. Louis, Mo.)

Because of the relative frequency of enchondroma, other benign lesions are often overlooked in the differential diagnosis of destructive lesions of the bones of the hand. Malignant lesions, both primary and secondary, are quite rare.

During a period in which 14 enchondromas of bones of the hand were encountered in the Washington University Department of Surgery, St. Louis, Missouri, 8 other destructive lesions of bones of the hand were found. Gout and rheumatoid arthritis were not included.

These 8 cases are reported, with reproductions of roentgenograms and histologic sections. They include 2 unclassified cases, 2 giant cell tumors, 2 hyperparathyroid cysts, 1 lipoma, and 1 inclusion cyst.—*Lois Cowan Collins, M.D.*

Willson, James K. V. The bone lesions of childhood leukemia; a survey of 140 cases. *Radiology*, May, 1959, *72*, 672–681. (Address:

Department of Radiology, The Johns Hopkins Hospital, Baltimore 5, Md.)

Long bone roentgen examinations in 140 cases of childhood leukemia were reviewed by the author. Included in this series are all cases of leukemia seen in the Harriet Lane Home with proof of the diagnosis by blood smear, marrow biopsy, or autopsy, and satisfactory roentgen-ray examinations of the long bones. The age range was from less than six months to thirteen years. There was no correlation between the presence of bone lesions and age, sex, race or cell type. In general, there was a correlation between duration of disease and bone changes.

The incidence of all bone lesions was 64 per cent, or 89 of 140 cases. The most common manifestation was a transverse line of radiolucency at the ends of the long bones, present in 76 cases. This narrow zone of decreased density, 1 to 6 mm. in width with a well defined proximal margin, is just beneath the metaphysis. The lateral cortical margins and the metaphyseal plate are usually intact, a point of differentiation from scurvy. Under the age of two years the transverse line of radiolucency is a nonspecific finding. It may be observed in a number of diseases. It may be seen not only in diseases which are unlike leukemia clinically but also in various infections which may resemble leukemia. Over the age of two years the transverse line of radiolucency is rarely seen in any other disease.

The next most common lesion was an osteolytic destructive process, present in 53 cases (38 per cent). This appears first in the spongiosa of the long bones and often shows symmetric involvement. With progression of the disease, destruction extends down into the shafts of the bones. Involvement may extend to the bones of the ankles and wrists and the tubular bones of the hands and feet, as well as the axial skeleton. The destructive lesions may be divided into three subtypes: (a) multiple punctate radiolucencies, (b) a decalcification of the metaphysis, and (c) frank destruction.

Periosteal elevation along the shafts of the long bones was evident in 27 cases (19 per cent). In 3 cases it was the only change. Pathologic fractures were present in 3 cases. Osteosclerosis was not observed.

When the bone lesions of leukemia are advanced, their appearance is usually characteristic. Advanced metastatic neuroblastoma may present a similar pattern. The transverse radiolucent metaphyseal line does not occur, however, and the differential diagnosis is usually clear, clinically. Roentgenologically, the presence of a transverse radiolucent metaphyseal line associated with destructive lesions is in general indicative of leukemia.—*Arno W. Sommer, M.D.*

Jacobson, Harold G., Fateh, Hassan, Shapiro, Jerome H., Spaet, Theodore H., and Poppel, Maxwell H. Agnogenic myeloid metaplasia. *Radiology*, May, 1959, *72*, 716–725. (Address: H. G. Jacobson, Montefiore Hospital, 210th St. and Bainbridge Ave., New York 67, N. Y.)

The authors present 20 cases of agnogenic myeloid metaplasia proved by bone biopsy or autopsy together with tabular summaries of clinical findings, blood picture, and sites of bone involvement. This disease is characterized by hematopoiesis occurring outside the usual marrow sites and is usually accompanied by fibrosis of the marrow. Synonyms include "pseudoleukemia with splenic-cachexia", "osteosclerosis with myelofibrosis", "splenic pseudoleukemia", "myeloproliferative syndrome", "myeloid metaplasia", and "mylofibrosis".

Roentgenologically, the thoracic cage, humeral shafts, and pelvic girdle were most frequently involved while the femoral shafts, dorsal and lumbar spine and shoulder girdle presented changes to a slightly less extent. There were two types of changes seen: first, a diffuse, overall increase in bone density, widespread and of varied intensity, and second, cases in which increased bone density was associated with variegated zones of radiolucent mottling or interspersed with linear and nodular opacities. The high frequency of involvement of the thoracic cage is stressed by the authors who believe the diagnosis may be suggested on viewing the chest roentgenogram.

The differential diagnosis includes such diseases as osteopetrosis, metastatic malignancy especially from the prostate or breast, fluorine sclerosis, congenital hemolytic anemia, and phosphorus poisoning.—*John S. Alexander, M.D.*

Fawcitt, John. (Manchester, England.) Some radiological aspects of congenital anomalies of the spine in childhood and infancy. *Proc. Roy. Soc. Med.*, May, 1959, *52*, 331–333.

Congenital anomalies of the spine are most frequently discovered by the radiologist and it is his responsibility to evaluate the significance of his finding. The author discusses chiefly two types of congenital anomalies of the spine: coronal cleft vertebrae and spina bifida of the lower lumbar and sacral spine.

He describes coronal cleft vertebrae and presents a suitable illustration. He notes that this deformity is probably more common than previously thought. He believes that the value of recognizing it lies mainly in the reassurance which can then be given to those concerned that it is only an anatomical variant and not necessarily pathologic. The cleft vertebrae are most frequently found in the later stages of pregnancy or soon after birth of the infant. Only a few have been followed for any length of time but the defect is said to disappear during very early life.

Very few statistical reviews of the incidence of spina bifida in childhood have been made. For this reason, the author reviews 500 roentgenograms of children made at Booth Hall Childrens Hospital in Manchester, England. Of these cases, 82 per cent showed some element of spina bifida occulta of varying degrees.

The cervical spine is prone to show errors of segmentation such as are seen in Klippel-Feil deformity. Hemivertebrae are most commonly seen in the thoracic spine. The lumbar spine and upper sacrum are most commonly associated with spina bifida. Abnormalities of the sacrum are usually those of aplasia or hypoplasia associated with disturbances of the urinary tract.

The author concludes that spina bifida and other minimal deformities should be reported by the radiologist.—*Richard E. Kinzer, M.D.*

Grainger, Ronald G. Procto-colitis and other pelvic infections in relation to ankylosing spondylitis; with a note on the vertebral venous system. *J. Fac. Radiologists*, July, 1959, *10*, 138–150. (From: St. Thomas's Hospital, London S.E. 1, England.)

The etiologic factors concerned in the pathogenesis of ankylosing spondylitis are still unknown. While this condition may be included in the rheumatoid or collagen group of diseases, the mere labeling of a disease pattern as a rheumatoid or collagen disease provides no direct help in assessing the etiologic or provocative factors. Little or nothing is established concerning any precipitating or exciting factors, which in a susceptible patient may stimulate the particular pattern of reaction of ankylosing spondylitis.

The author reports the coincidence in 7 patients of ankylosing spondylitis and ulcerative colitis. His paper is designed as a provocative review of the possible influence which infection within the pelvic cavity may exert as an exciting factor in the development of ankylosing spondylitis.

Unilateral and bilateral nonsuppurative, destructive, sacroiliac arthritis is a well recognized feature of Reiter's syndrome, nonspecific urethritis (incomplete Reiter's syndrome), gonorrhea, proctocolitis and paraplegias. In all of these conditions there exists a focus of chronic pelvic sepsis. There is no certain clinical or roentgenologic distinction of this arthritis from that which almost invariably is the first manifestation of classic ankylosing spondylitis. These patients with known chronic pelvic infection may develop clinical and roentgenologic evidence of pelvic and spinal osteopathic, arthritic and ligamentous changes indistinguishable from those found in classic ankylosing spondylitis.

The evidence available regarding the association of pelvic infection and sacroiliac arthritis is twofold. On the one hand it is known that patients with a known focus of chronic pelvic (genitourinary or alimentary) sepsis may develop destructive, nonsuppurative sacroiliac arthritis. On the other hand, it has recently been confirmed that 80 per cent to 90 per cent of patients with ankylosing spondylitis have a chronic prostatovesiculitis as opposed to a controlled series with a 33 per cent incidence.

The author suggests that intrapelvic infection, usually prostatovesiculitis but occasionally rectosigmoiditis may cause a bilateral, nonsuppurative, destructive sacroiliac arthritis. In patients with the necessary genetic and constitutional makeup, this arthritis may be the first manifestation of that particular pattern of patient reaction which is called "ankylosing spondylitis."

The vertebral venous system may play a prominent role in this pelvic infection—ankylosing spondylitis relationship. Batson has described the interesting history of the discovery of this system. In man and the majority of mammals there exists a vertebral venous system consisting of two main longitudinal venous sinuses, freely communicating with each other, having very thin walls and no valves, and usually containing a small quantity of venous blood under low pressure. In the pelvis, sacral branches of the internal iliac veins which drain the pelvic viscera pass through the anterior sacral foramina to anastomose with the vertebral plexus. There is much clinical evidence favoring the existence of a preformed pathway from the pelvic viscera to the spinal column, sacrum and pelvic bones. A vertebral venous plexus that communicates freely with the pelvic visceral veins can be demonstrated anatomically by dissection and by experimental injection. It is reasonable to suppose that this vertebral venous system is the route of bacterial embolism, which may explain the occasional occurrence of spinal and pelvic osteomyelitis following pelvic sepsis. It may be also the route taken by pelvic sepsis acting as a possible provocative factor in ankylosing spondylitis.—*Samuel G. Henderson, M.D.*

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